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Journal of the Medical Society of New Jersey

Platelets and Hypertension
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Giant Adenoma of Rectum
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Malignant Ventricular Impulses
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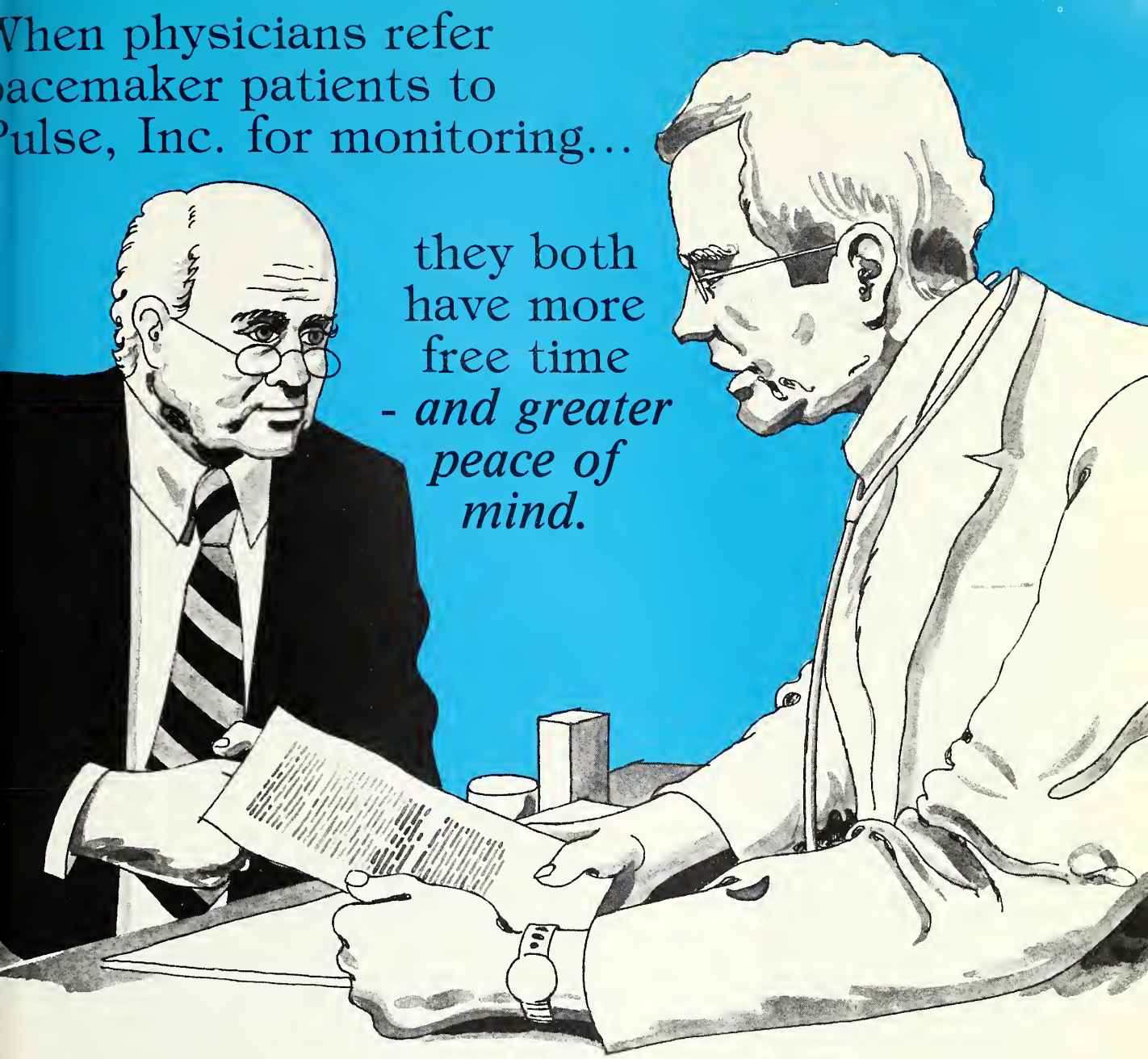
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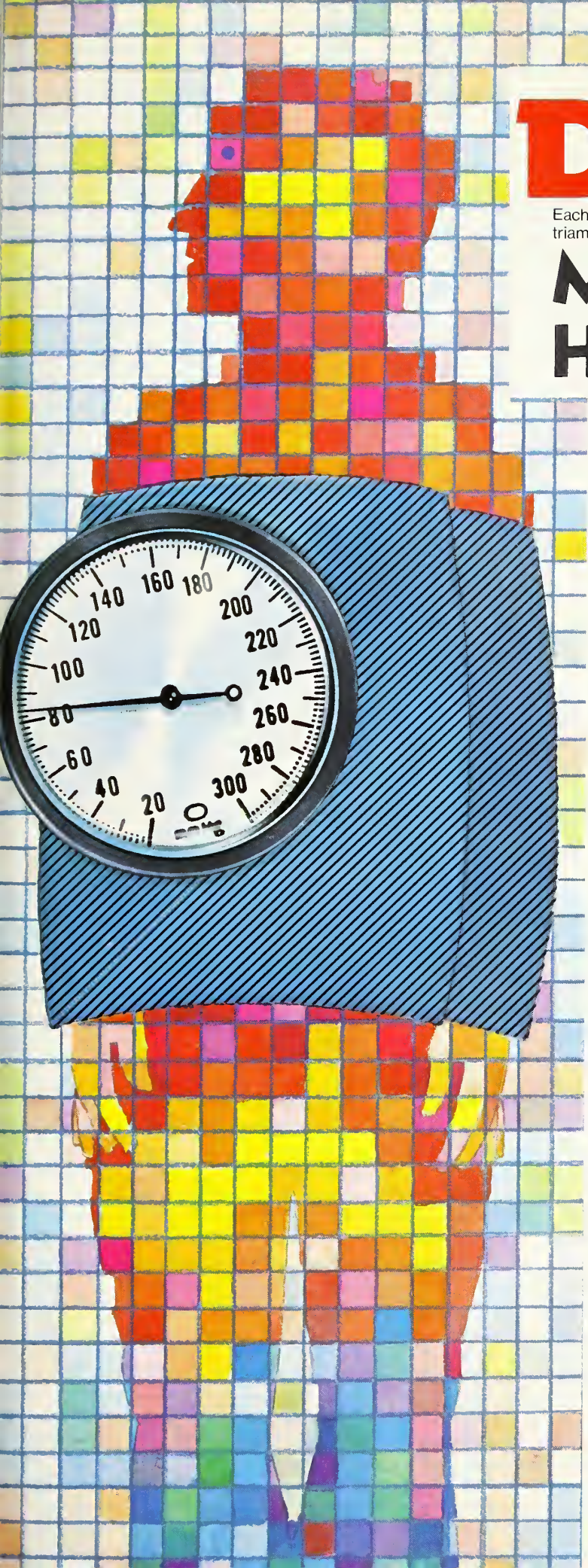
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Cover Photo

The cover photograph of Crow's Wood Nature Center, Haddonfield, taken in the winter of 1978, was provided by John L. Krause, Jr., M.D., plastic surgeon from Cherry Hill and a member of the Camden County Medical Society. He is a medical and nature photographer as well as lecturer on these subjects, and has supplied us with cover material in the past, most recently in July 1978.

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Usage in Pregnancy: Although theophylline has been used for many years, with no evidence of adverse fetal effect or teratogenicity, its safety in pregnancy has not been established. Therefore, use of Bronkodyl during lactation or in women of childbearing potential requires that possible benefits of the drug be weighed against possible hazards to fetus or child.

Precautions: Bronkodyl should be used with caution in patients with cardiac or circulatory disease.

Adverse Reactions: *Gastrointestinal:* epigastric distress, nausea, vomiting. *Cardiovascular:* palpitations. *CNS:* insomnia, restlessness, irritability, convulsion.

Dosage and Administration: *Adults:* Usual dosage of Bronkodyl is 200 mg. every 6 hours (four doses in each 24 hours). This dosage may be adjusted to reflect individual clinical response as an indication of slow or rapid metabolism of the drug. If adverse reactions are encountered, each dose may be reduced, or the interval between doses may be lengthened, or both. If clinical response is not satisfactory, indicating possible rapid inactivation of the drug, dosage may be gradually increased to achieve the desired response. In some instances of either too slow or too rapid metabolism, plasma levels of theophylline should be determined and dosage adjusted accordingly to achieve levels above 10 mcg./ml., but not to exceed 20 mcg./ml.

Dosage in Children: Usual dosage should be based on administration of 10 mg. per kg. per 24 hours, divided in 4 doses per day, given every 6 hours. As this may not be possible with use of the capsules, Bronkodyl elixir may be used. Theophylline saliva levels (approximately 60% of simultaneous blood levels), may facilitate dosage adjustments, especially in children, to obtain appropriate response.

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[1] Tinkelman, D.G., Carroll, M.S., Vanderpool, G., Jones, M.: The bioavailability of theophylline in elixir and micro-pulverized forms. Medical Challenge 10:24-26, 1978.

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*The safety and effectiveness of Motrin have not been established in patients with Functional Class IV rheumatoid arthritis (incapacitated, largely or wholly bedridden, or confined to wheelchair; little or no self-care).

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Motrin 400 mg TABLETS
ibuprofen, Upjohn

The confidence that comes from experience—one more reason to prescribe Motrin.

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The confidence that comes from experience—
one more reason to prescribe

Motrin 400 mg TABLETS
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Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin (ibuprofen) is gastrointestinal (4% to 16%). This includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness*, headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

Incidence: Unmarked 1% to 3%; *3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Suggested dosage is 300 or 400 mg t.i.d. or q.i.d. Do not exceed 2400 mg per day.

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Unit-dose package of 100	NDC 0009-0750-06
Unit of Use bottles of 120	NDC 0009-0750-26

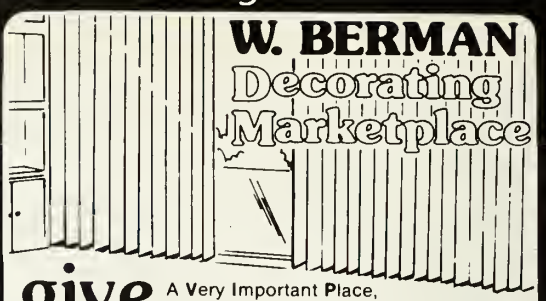
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EDITORIALS

Postgraduate Medical Education in New Jersey: A Challenge

The major problems faced by New Jersey hospitals with housestaff training programs relating to the implementation of Public Law 94-484 clearly are documented in the paper by Dr. Harvey Hechel in this *Journal* (page 49). In this law, Congress declared that "there is no longer an insufficient number of physicians and surgeons in the United States, and that there is no further need for affording preference to alien physicians and surgeons in admission to the United States." The law is complex and addresses many issues, but specifically it affects housestaff programs. Foreign physicians no longer may enter the United States as exchange visitors (J-Visa) unless they have passed an equivalent of the National Board of Medical Examiners' examination, Parts I and II (Visa Qualifying Examination—VQE). While these requirements may be waived until December 31, 1980, if there is proof of substantial disruption in health services, restrictions on this waiver substantially will limit the recruitment of foreign housestaff even before 1981.

Dr. Hechel points out that New Jersey has the highest percentage of foreign medical graduates (FMG) in the United States, although New York State may have even greater problems because they have four times the number of FMGs in their training programs. Those of us concerned with both clinical care and medical education in the State of New Jersey should come to grips with the implications of these statistics. Several of the realities pointed out by Dr. Hechel cannot be altered. We neither can move New Jersey south of the Mason/Dixon Line, nor can we move New York City or Philadelphia into this State. We must, however, examine whether our housestaff training is of high enough quality to attract bright young physicians despite the demographic trends. All too often, housestaff, particularly foreign housestaff, has been used as "cheap labor," and physicians have put pressure on their hospital administration to attract housestaff programs in order to relieve them of hospital chores and night call. We must realize that housestaff personnel comes to us with an unwritten "social contract." This contract states that, in return for relieving faculty and physicians of many of their time-consuming responsibilities, they will receive intensive training in both clinical and scientific aspects of medicine. In good programs, the trade-off should be at least equal in time, with even a negative balance in the best programs.

Undoubtedly, some of our borderline training programs will die in the near future. However, not all of the statistics should lead to pessimism. Quite to the contrary, FMGs in New Jersey residency programs have fallen by 16 percent in the seven years from 1970 to 1977. An increasing number of these FMGs represent New Jersey residents who return *via* the Fifth Pathway program. The College of Medicine and Dentistry of New Jersey currently is graduating one hundred and eighty new physicians annually and this number is likely to be doubled by 1985. However, these numbers should not divert us from the lesson to be learned from Dr. Hechel's statistics, and that is that we must reexamine our teaching programs and attempt in every way to make them of such excellence that the best and brightest of our students will remain for training and practice in the State.

Norman H. Ertel, M.D.

Professional Liability

New Jersey physicians practice in a much healthier professional liability environment today as compared to a few years ago for at least two good reasons:
The *Peer Review Program of the New Jersey State Medical Underwriters, Inc.* (the attorney-in-fact of the Medical Inter-Insurance Exchange of New Jersey) and the new *Supreme Court Rule on Professional Liability Subpanels.* It behooves all of us to be cognizant of these entities and to participate in each as peer reviewers and panel members.

Peer Review Program

The first annual update on the results of the Peer Review Program, as reported to the Board of Trustees, showed some striking and gratifying statistics. Of the 479 cases reported, 94 were closed and of these, 45 were peer-reviewed. Of the 385 outstanding cases, 111 were peer-reviewed. Of those which were peer-reviewed, 48 percent were deemed indefensible while 52 percent were defensible. Of the 45 peer-reviewed cases *closed,* eight went to suit while 47 of the 111 peer-reviewed *outstanding* cases went to suit.

The dollar values of the indemnity figures and expenses are of great interest. Of the 45 peer-reviewed closed cases, no indemnity dollars were paid for the defensible cases while expenses amounted to only $374.50. Indemnity paid to ten indefensible cases totaled $410,300 with expenses of $1,799.60. Thirty closed cases in which formal peer review was waived resulted in indemnity payments which averaged $2,024.88 per case with expenses of $5.73.

Such dollar amounts are a far cry from the six and seven-figure settlements and judgments which newspaper editors

love to make into headlines. Even more striking is the fact that the average number of days from the date the cases were reported to the date they were closed was only 120.

Professional Liability Subpanels

Effective with the September 11, 1978 New Jersey Supreme Court term, an amended Rule 4:21 became effective. Known as *Professional Liability Claims Against Members of the Medical Profession: Procedure,* the amended rule is a much improved attempt to serve "the common interest of the public and the medical and legal professions in the processing of medical malpractice actions."

Its ultimate aims are "to discourage baseless actions and to encourage settlement of those actions based on reasonable medical probability; to monitor efficiently these cases through the court; and to assist in the early disposition of medical malpractice." (see page 60 for complete text.)

The Medical Society of New Jersey, the New Jersey Judiciary, and the Bar Association believe that the Rule takes a giant step forward in the struggle to control the cost of professional liability insurance, while providing an opportunity for fair compensation in a timely fashion to the person with a valid claim of injury. The essence of its success will be the ability of the procedure to reduce or eliminate non-meritorious or frivolous claims while encouraging settlement of meritorious claims without the need for trial. Thus, reduced expenses and shortened lag-time from claim to closure will be in the best interest of all concerned.

Amended Rule 4:21 specifically provides that the court shall conduct a mandatory pretrial conference for every medical malpractice suit "not later than twelve months following service of process upon the defendants." The panel which hears the case shall consist of a judge who presides as chairman, a physician, and an attorney. The doctor, who shall be selected from a panel of physicians designated by the Medical Society of New Jersey or a panel designated by the New Jersey Society of Osteopathic Physicians and Surgeons, will be from the discipline of the defendant doctor or doctors and, where practicable, *he or she will be a practitioner in the field of specialty involved in the case.* The attorney is selected from a list provided by the New Jersey Supreme Court.

The hearing is informal and without a stenographic record. The parties to the suit set forth their factual and legal contentions to the panel, which may request an additional expert medical witness from the specialty involved to assist it in its determination. Such an expert medical witness shall be paid a fee and expenses by the parties to the hearing, although the panel physician and the panel attorney serve voluntarily and without compensation. The medical expert witness and the doctor member of the panel may be called to testify at a subsequent trial by any of the parties, who shall be responsible for the fees and expenses of the doctors for such testimony.

During the hearing, the panel shall evaluate the facts as to each medical issue and determine whether the claim is based on reasonable medical probability. If the decision is unanimous, the findings shall be admissible in evidence at the trial at the request of any party to the hearing and, although the recommendation is not binding upon the jury or upon the judge, it shall be given such weight as they choose to ascribe to it in the light of all the relevant evidence adduced at the trial. It would appear that such a recommendation would be given serious consideration by the very nature of the entire procedure.

The panel judge, who is assigned by the Assignment Judge in each county, shall fix the time and place of the pretrial hearing but, to the extent possible, it will take place in the home county of the panel members on a date acceptable to the members and to the parties to the suit.

Thus it appears that a New Jersey physician who is facing a medical malpractice claim has two eminently fair procedures available prior to an actual trial. Both the Peer Review Program and the Mandatory Pretrial Subpanel Hearings ought to serve the desirable purposes for which they were developed. To be effective, a large enough panel of physicians, particularly specialists, must be available. Approximately 1500 physicians in New Jersey have volunteered to participate in the peer review mechanism, while 200 of them actually have participated to date. It is essential that sufficient volunteer doctors in every county make themselves available for service for either peer review or pretrial hearings. It will help all of us both individually and collectively to control the threat of frivolous claims and to constrain the rampant costs of medical liability insurance premiums. We must not rest on our laurels of the past year, but must continue to participate actively in these extremely valuable processes. A.K.

Is Your License Current?

It seems that there are physicians in New Jersey technically practicing without a license. The cause is a computer failure, not any wrongdoing on the part of such practitioners.

In 1971, a law requiring registration of all physicians went into effect. This law provides for automatic suspension of the license of a physician who does not hold a current paid-up registration. The current period is July 1, 1977 to June 30, 1979.

Any physician who has failed to submit his registration fees in the past may have his license reinstated by the Board of Medical Examiners retroactively to the period of time when he originally had failed to pay his registration. All he must do is pay past registration fees and a $25 reinstatement fee to the board.

If you do not have a current registration certificate, write (or phone) *quickly* to: Alfred J. Schuster, Executive Secretary, State Board of Medical Examiners, 28 West State Street, Trenton, New Jersey 08608. A.K.

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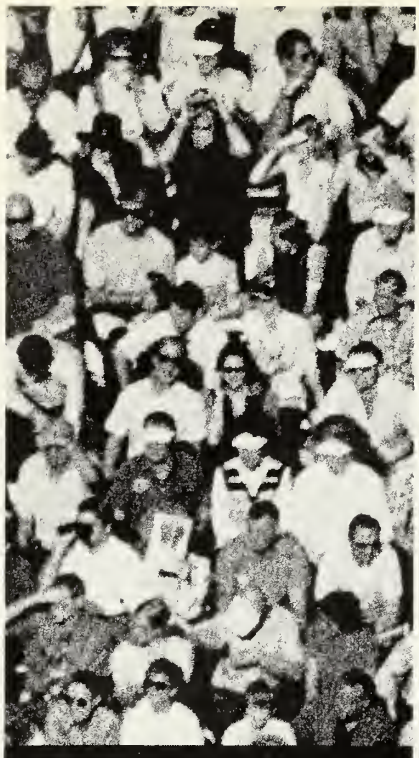


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Platelet Function and Hypertension in Humans*

ALAN I. FLEISCHMAN, Ph.D., Montclair
HANS-U. COMBERG, M.D., and
CURTIS G. HAMES, M.D., Claxton, Georgia

In clinically normal subjects platelet aggregation tendency was found to be related to blood pressure. Untreated hypertensive patients had a significantly greater aggregation tendency than did patients with normal blood pressure or patients with hypertension who are receiving drug therapy.

In an earlier report[1] anticipatory stress of minor surgery was shown to increase both blood pressure and platelet aggregability in humans. Within a given individual, changes in blood pressure could be correlated with changes in platelet aggregability in both normal subjects and in hospitalized neurological patients.[2] Van Kessel *et al.*[3] found that platelet aggregability is increased significantly in patients with hypertension and pregnancy as compared to pregnant women with normal blood pressure.

Hypertension is considered a risk factor in both cardiovascular and cerebrovascular disease.[4-9] Haerem[10] reported the presence of platelet aggregates in intra-myocardial vessels in cases of sudden death from coronary artery disease. Both Sullivan *et al.*[11] and Fleischman *et al.*[12] noted an increase in platelet adhesiveness in patients with coronary artery disease. Walsh and co-workers[13] noted increased platelet coagulant activity in patients with transient cerebral ischemia.

Since hypertension is a known risk factor and since there are reports that patients under stress and hypertensive individuals exhibit increased platelet aggregability, studies were undertaken to evaluate the effect of hypertension on platelet aggregability and to study the effect of blood-pressure lowering by usually employed drugs upon platelet aggregation.

MATERIALS AND METHODS

In the first study, conducted in northern New Jersey, the patient population consisted of 105 normotensive, 37 untreated hypertensive, and 75 treated and controlled hypertensive patients. In study two, conducted in Evans County, Georgia, the patient population consisted of 40 normotensive, 26 treated and controlled hypertensive, 13 treated hypertensive who had not responded to the medication, and ten untreated hypertensive individuals. The patient population varied in age from 35 to 85 years and was distributed approximately equally between male and female. No patient in this study was known to suffer from any disease, chronic or acute, other than hypertension, except for one patient in the study two who was an insulin-controlled diabetic.

In both studies hypertension was defined as a diastolic blood pressure above 90 mmHg on repetitive measurements at rest. The drugs used in this study were those prescribed by the individual patient's treating physician. In study one, 82 percent were treated with diuretics, primarily thiazides or furosemide, six percent were treated with a β-blocker, and two percent each were on α-methyldopa or reserpine respectively. The remaining ten percent were treated by combina-

*This study was supported in part by a grant from the Charles Edison Fund and by grant number USPHS 3341 from The National Heart, Lung, and Blood Institute. The opinions expressed in this paper are those of the authors and are not necessarily the official position of the New Jersey State Department of Health. Dr. Fleischman may be addressed at the Division of Community Health Services, New Jersey State Department of Health, 48 Plymouth Street, Montclair 07042. Drs. Comberg and Hames are with the Evans County Health Department Heart Research Project, Claxton, Georgia 30417.

Table 1
Comparison of Blood Pressures and Platelet Function in Normotensives, Treated Responsive Hypertensives, and Untreated Hypertensives in Study 1, Northern New Jersey

Group	Number of Individuals	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)	Aggregation Time (Seconds)	% Failing to Disaggregate
Normotensives	105	124±1.31	76±0.8	341±24	11.4
Treated hypertensives	75	153±2.5^{xx}	89±1.4^{xx}	302±3.9	13.3
Untreated hypertensives	37	162±2.6^{xx}	91±1.9^{xx}	252±38^{xx}	21.6^{x}

1. Mean± standard error of mean
xx $p<0.01$ compared to normotensives
x $p<0.05$ compared to normotensives

tions of drugs, primarily a thiazide and either a -methyldopa, or rauwolfia derivative. In study two, 33 percent were treated with diuretics, five percent with guanethidine and 62 percent were on combination therapy. The patients had been on therapy for periods of at least one year and in many cases over ten years. Except for the drugs employed in the treatment of hypertension, no agents known to affect platelet function were taken by any of the patients.

The aggregation of platelets in flowing blood, in the presence of all other formed elements of the blood, was accomplished by drawing blood via a 19-gauge thin wall siliconized butterfly needle, from an antecubital vein through a microfilter of 20 micron pore size into a motor driven syringe at a constant rate of 2.0 ml per minute. This filtration method was based on the use of a pore size which permitted the passage of red and white cells and platelets but was occluded by platelet aggregates. The entire system was siliconized and maintained at 37°C. Heparin was infused into the system proximal to the filter at a concentration of 25 IU per ml blood. Pressure was monitored proximal and distal to the filter, the rise in pressure difference reflecting occlusion of the filter by platelet aggregates. The aggregation time was defined as the time in seconds required to reach a pressure differential across the filter of five mmHg. Following aggregation, the heparin infusion was discontinued and a 38 percent sodium citrate solution was infused at a rate of 0.2 ml per minute. Disaggregation time was defined as the time in seconds required to clear the filter pores as shown by a reversal of the pressure gradient. To check the patency of the needle, the pressure between the needle and the filter was continuously monitored.[2]

In order to evaluate the diet of the patients, the fatty acid composition of the serum triglycerides was determined by gas liquid chromatography.[14] Serum lipids were evaluated by semi-automated methodologies.[15] Hematology was done by standard laboratory methodology. Statistical analysis was performed on the data by the student "t" test for aggregation and the chi square test for percentage disaggregation.[16]

RESULTS

In preliminary studies, no sex or age-related differentiation in platelet function was noted. The reference range for aggregation time in clinically normal individuals calculated by the method of Chu, Cheung, and Turkington[17] was 195 to 545 seconds in age range between 15 and 85 years.

Examination of serum lipid values for this population indicated that approximately 30 percent were hyperlipidemic. Of these, six percent were hypercholesterolemic, 16 percent were hypertriglyceridemic, and the remaining eight percent were mixed hyperlipidemics. The distribution in the control groups was similar. No significant difference was found between the various groups in hemoglobin and hematocrit.

In the first study (Table 1) the mean systolic blood pressure for the 105 normotensives, for 75 treated and responsive hypertensives, and for 37 untreated hypertensives differed from each other at the 99 percent confidence level. A similar significant difference was noted in the diastolic blood pressures between the three groups, $p<0.01$. No significant difference in aggregation time was noted between the normotensive and the treated and responsive hypertensives. The difference between either the normotensives or the treated responsive hypertensives and the untreated hypertensive was significant at $p<0.01$.

In examining the effect of hypertension upon disaggregation time, it was found that some individuals in all groups failed to disaggregate in 600 seconds. This maximum time was chosen so as to limit the time an individual was connected up to the instrument and by the limitations imposed by the 50 ml syringe employed for blood drawing. Thus, in examining these data, the percentage of persons in the three groups failing to disaggregate was evaluated. The participants in the untreated hypertensive group had a greater percentage failing to disaggregate ($p<0.05$, Table 1), than either of the other two groups.

The results of the second study are shown in Table 2. Both systolic and diastolic blood pressures were greater in either the untreated or the treated but uncontrolled hypertensives than in either the normotensives or the treated and controlled hypertensives, $p<0.01$. No significant differences were noted in disaggregation between the groups. This appears to be related to the smaller number of untreated hypertensives in this study.

As in previous study, no significant difference in aggregation time was noted between normotensives and either treated group of hypertensives. The untreated hypertensives have significantly shorter aggregation time than either the normotensives or the treated and controlled hypertensives, $p<0.01$, and also the treated but uncontrolled hypertensives, $p<0.05$.

The aggregation times in Evans County, Georgia study II were significantly shorter than those found in northern New Jersey study I. Linoleic acid concentrations in the diet were evaluated in both areas by gas liquid chromatographic analysis of the serum triglyceride fatty acids.[14] The mean dietary linoleate as calculated from these data in Evans County, Georgia was 2.10±0.66 percent of calories while in New Jersey it was 3.2±0.10 percent of calories. Fleischman

Table 2
Comparison of Blood Pressures and Platelet Function in Normotensives, Treated Controlled Hypertensives, Treated Uncontrolled, and Untreated Hypertensives in Study 2, Evans County, Georgia

Group	Number of Individuals	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)	Aggregation Time (Seconds)	% Failing to Disaggregate
Normotensives	40	122±2.24[1]	76±1.65	255±18.36	15.0
Treated controlled hypertensives	26	126±2.96	80±0.97	233±19.15	15.4
Treated not controlled hypertensives	13	149±7.31[a,d]	98±1.33[a,d]	211±27.64[c]	15.3
Untreated hypertensives	10	152±6.30[a,d]	99±0.82[a,d]	163±25.94[a,b]	10,0

1. Mean± standard error of mean
a $p<0.01$ compared to normal
b $p<0.01$ compared to responsive hypertensives
c $p<0.05$ compared to untreated hypertensives
d $p<0.01$ compared to treated controlled hypertensives

et al.[18] reported that increasing dietary linoleate increased the time required for platelets to aggregate and this would appear to explain, at least partly, the differences found in the mean values of aggregation time in the two areas.

DISCUSSION

The increase in aggregation tendency found in untreated hypertensive subjects agrees with the data of Van Kessel *et al.*[3] It was not totally unexpected that hypertensive subjects would exhibit increased platelet aggregability.[1] Platelet function is reported to be controlled at least partially by prostaglandins and thromboxanes.[19,20] Similarly these hormones are involved in the regulation of arterial pressure[20-22] and affect the renin-angiotension system.[20-23] Those agents which alter hypertension might control the synthesis and metabolism of prostaglandins. Taube *et al.* have found this to be true for clonidine, α-methyldopa and reserpine.[24] Triebe and Block reported that increased linoleic acid could counteract the effect of salt-induced hypertension in rats.[36] Ten Hoor and van deGraaf found that the prostaglandin antagonist, acetylsalicylic acid, counteracted the effect of increased linoleic acid in reducing sodium chloride-induced hypertension in rats.[37] The improvement of platelet function by anti-hypertensive agents noted in this study would be in accord with the data of Taube *et al.*[24]

Not all reports indicate that stress, which might be equated with elevated blood pressure, negatively affects platelet function. In 1975, Haft and Arkel reported, in a study of hospital interns and residents, that the application of a severe stressor, grand rounds, decreased platelet aggregability.[25] These results are similar to those reported by O'Brien *et al.* who noted, in a study of patients undergoing major surgery, that platelets could become refractory and postulated that this might be due to exposure of the platelets to high circulating ADP levels during the surgical trauma. [26] Haft states that their findings might be explained by a similar mechanism; i.e., the platelets became refractory due to persistent exposure to high levels of catecholamine.[27] Haft also noted that young platelets are aggregated more easily than old platelets and that it is possible that the young platelets in the circulation had aggregated intravascularly, sequestering in various capillary beds.[27] Thus, Haft and Arkel may have been studying older platelets that were less sensitive to aggregating agents.[25]

The Filtragometer employs heparin as an anticlotting agent in measuring platelet function. There have been a series of reports which have noted a mild thrombocytopenia associated with heparin coagulation.[28-31] Some severe cases have been reported also.[32-34] There is a question as to whether, in man, heparin induced platelet aggregation by acting on the platelets or by acting on some other system which then induced thrombocytopenia. In 1976, this question was resolved by Babcock *et al.* who noted that, *in vivo* in man, heparin activates an immune mechanism with the production of an antiplatelet antibody.[35] This antiplatelet antibody then reacts with the platelets inducing the thrombocytopenia. Thus, in the Filtragometer procedure, since the heparin is employed *in vitro,* the immune system cannot be activated with the production of antiplatelet antibody. In addition, since the time between the addition of the heparin and the making of the measurement is seconds, there is not sufficient time for significant antibody production.

Therefore, agents exhibiting a hypotensive effect might be expected to decrease the elevated platelet aggregation tendency noted in hypertensive subjects and conversely those agents which improve both aggregation and disaggregation of platelets might have a hypotensive action provided they do not completely inhibit all prostaglandin metabolism and synthesis. As it has been reported that increased linoleic acid will decrease both platelet aggregability and hypertension, it would appear that both conditions may be related to depressed or changed prostaglandin biosynthesis.[37-39] Since there appears to be an increased platelet aggregation tendency in hypertensives and also in patients with cerebrovascular disease,[12,13] it is possible that one association between hypertension and cerebrovascular disease can be through increased platelet aggregation tendency.

SUMMARY

The platelets of untreated hypertensive humans aggregated significantly more rapidly and disaggregated less frequently than those of either normotensive or treated hypertensive adults. In a study conducted in northern New Jersey on 105 normotensives, 37 untreated hypertensives, and 75 treated and controlled hypertensives, the platelets of the untreated hypertensives aggregated 26 percent faster than those of the normotensives and 16.6 percent faster than those of the treated patients. In the Evans County, Georgia study, the platelets of the untreated hypertensive patients aggregated 36 percent faster than those of the normotensives. The platelets

in all subclasses were less aggregable in the northern New Jersey cohort than in the Evans County, Georgia cohort. This appeared to be associated with an increased dietary linoleic acid intake in the northern New Jersey cohort. Since increased platelet aggregation tendency is associated both with hypertension and with cerebrovascular disease, a possible association between hypertension and cerebrovascular disease could be through increased platelet aggregation tendency.

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Diagnosis and Management of Asthma Part II: Drug Therapy in the Office

FREDRIC A. SCHULANER, M.D., Westfield and
MICHAEL S. MATTIKOW, M.D., Wayne

The prophylactic use of drugs has benefited the asthmatic considerably. Theophylline in therapeutic doses is the drug of choice, with sympathomimetics added separately when necessary. In selected patients cromolyn sodium has given excellent results, with beclomethasone by inhalation often negating the need for systemic steroids.

Drug therapy is an important part of the overall treatment program of the asthmatic. Primarily, the patient should try to avoid those immunologic and non-immunologic factors that are triggers of his asthmatic attacks. Since complete avoidance often is impossible or impractical, a drug regimen should be tailored to the individual patient. The type and frequency of medication depends on several factors.

One, is the *frequency* of attacks: some patients wheeze rarely; others never seem to stop. Another important factor is the *severity* or intensity of the episode. Some attacks are so mild that wheezing is hard to find on auscultation and only may be demonstrated by pulmonary function studies, while other attacks are so severe as to require emergency medication or even hospitalization. Also, the *sensitivity* of the patient to various medications will influence both the drugs used and the dosages employed.

THEOPHYLLINES

The first group of drugs to be tried in asthmatic patients are the theophyllines. Although this drug formerly was used to stop wheezing that already was present, a new philosophy has emerged, which prescribes the use of round-the-clock theophylline as prophylaxis.

This drug is both safe and effective when a blood level of 10 to 20 micrograms per ml. is maintained.[1] However, there are individual differences in the metabolism of theophylline, so that the optimum dosage may vary. Generally, younger patients require higher dosages per kilogram of body weight. Table I is a safe guide to the starting dose. Using this schedule, only a rare patient will have blood levels over 20 micrograms per mil—that level where toxic side effects begin to appear. Most patients will be in the therapeutic range, while those who have tolerated the drug but have not had a good clinical response can have their dosage increased slowly and carefully at three-day intervals. The availability of measuring serum theophylline levels at several New Jersey hospitals has made the use of this drug safer and more effective. The time to draw the blood is at the peak absorption, which for most liquids and uncoated tablets is approximately two hours after the drug has been taken; blood also should be taken at trough levels—six hours later. A level below therapeutic range would indicate a rapid metabolizer and the dose of theophylline should be increased cautiously.[2] In those patients whose wheezing persists, despite adequate blood levels, other medications must be added to the treatment program.

We suggest adding the second bronchodilator as a separate drug rather than using a fixed-dose proprietary preparation. This may allow one to build the second drug to the thera-

*This is the second in a series of three papers from the New Jersey Allergy Society, on the modern approach to diagnosis, office management, and hospital care of the asthmatic.

Toble 1
Storting Dose of Theophylline

	Younger Children	Older Children	Adults
Theophylline mg/kg/6hr	5.6	4.6	3.1
Aminophyllin (orol) mg/kg/6hr	7.0	5.8	3.9
Aminophyllin (IV infusion) mg/kg/hr	1.1	0.96	0.65

Table 2
Theophylline Preporotions

Proprietory Name	Form	Mg. of Theophylline	Frequency
Aminophylline	Tablets	160, 80	q6h
Slophylline Gyrocops	Capsules	250, 125, 60	q8h
Slophyllin Toblets	Toblets	200, 100	q6h
Slophyllin 80	Syrup	80 mg/15 ml.	q6h
Slophyllin GG	Syrup	150 mg/15 mi. (Guaifenesin 90 mg.)	q6h
Elixophyllin	Elixir	80 mg./15 ml.	q6h
Elixophyllin	Capsules	200, 100	q8h
Somophyllin	Orol Liquid	90 mg./5 ml.	q6h
Theo-Dur	Toblets	300, 100	q12h
Theophyl 225	Toblets	225	q6h
Theophyl SR	Copsules	250, 125	q8h
Theophyl 225	Elixir	225 mg./30 ml.	q6h

peutic blood levels before toxic levels of theophylline are reached.

The bio-availability of many preparations differs, so one must know the actual theophylline content of each drug used. For example, aminophylline contains 80 percent theophylline, while a 200 mg. tablet of Choledyl® has only 128 mg. of usable theophylline. Recently there has been a great increase both in the number of preparations and dosage forms of theophylline products. It is impossible to know them all, so the most practical solution is to know a few thoroughly. One advantage of the newer drugs is that the amount of theophylline in the preparation is clearly stated. Table II gives several examaples of preparations, the amount of drug available, and the frequency of use.

Acute infections, troleandomycin, erythromycin, heart failure, and liver disease can slow theophylline elimination; accumulation of the drug can produce toxicity at modest doses. Metabolism of theophylline appears to remain stable over long periods of time; however, there are exceptions so blood levels have to be repeated in patients who have increased symptoms or toxic effects.

SYMPATHOMIMETICS

The sympathomimetics are indicated as an alternative to theophylline in patients intolerant to several different forms of that class. It would be used in addition to theophylline, if the patient is on full therapeutic dosage, as judged by serum theophylline levels, and is still having significant wheezing. Ephedrine is the original oral form that has been used for many years, but today there are several newer preparations which theoretically have more $beta_2$ sympathetic activity, less central nervous system side effects, and longer duration of action. These newer drugs, however, are expensive and probably should be reserved for those patients who get side effects from ephedrine before therapeutic results are achieved.[3] Table III lists these oral sympathomimetic agents.

Sympathomimetic sprays really are not indicated for continual use, but they may be effective in older children, adolescents, and adults for acute episodes of wheezing and dyspnea. However, there are several potential problems that have been associated with their use. First, careful explanation must be given in the mechanics of proper operation. In the severe asthmatic, the inhalent often is ineffective and may be harmful to patients who do not seek medical advice, but continue to use the drug despite the fact that the bronchospasm is becoming more and more severe. An occasional patient will have an idiosyncratic reaction, with actual exacerbation of wheezing after its use. Therefore, the physician carefully must explain the appropriate use of these medications so they will be effective and safe.

Metaproterenol is one of the newer additions to these aerosol sprays. It has more specific $beta_2$ sympathomimetic activity than any of the others, and can be given orally. However, in the future, even more specific and potentially more active drugs will become available

CROMOLYN SODIUM

Cromolyn sodium, which was introduced several years ago for the treatment of asthma, has an unique mechanism of action. The drug stabilizes the mast cell membrane, thus preventing the release of histamine and other mediators.[4] It has no bronchodilating effects or any antagonistic action on the chemical mediators of asthma. The dosage is one capsule via inhalation, four times a day, regardless of age or body weight. If patients do well, this dosage often may be decreased. Its effects are prophylactic only, and cromolyn sodium has no place in the treatment of acute attacks of asthma. Although major side effects are rare, many patients will complain of dryness of the mouth and transient sore throat. Less common is slight wheezing and chest tightness immediately following inhalation of cromolyn.

Cromolyn sodium has been found to be useful in several circumstances, the most important is in the prophylaxis of severe perennial asthma. The drug also can give temporary protection against air-borne allergens such as seasonal pollen or animal dander. Although it can be used in exercise

Table 3
Oral Sympathamimetic Drugs

Drug	Form	Dose	Frequency
Ephedrine	Capsule 25 mg.	0.25 – 0.50 mg./Kg	q4h
Ephedrine	NF Syrup 20 mg./5 ml.		
Metapraterenal	Tablet 10, 20 mg.	10 – 20 mg.	tid
	Syrup 10 mg./5 ml.	6 – 9 yrs. 5 ml. (under 60 lbs.)	tid
Terbutaline	Tablets 2.5, 5 mg.	2.5 – 5 mg.	tid

induced bronchospasm, other forms of therapy, such as theophylline, are less expensive and probably more effective. Generally, cromolyn is most beneficial in children and adolescents with perennial extrinsic asthma. The drug should be tried by all patients whose asthma cannot be controlled adequately by round-the-clock bronchodilator therapy, before initiating prolonged corticosteroid treatment. One must give the patient at least a one-month's trial; if there is no improvement, then further cromolyn therapy is not likely to help. One can expect approximately sixty percent improvement of patients with extrinsic asthma on cromolyn therapy. However, recent reports suggest that with theophylline treatment, where therapeutic levels are maintained, one can achieve even better results than with the use of cromolyn alone.[5]

CORTICOSTEROIDS

In that small group of patients still wheezing in spite of theophylline, sympathomimetics, and cromolyn sodium, corticosteroids may be required. Generally, a short course (three to five days) of prednisone in dosage of 20 to 45 mg. per day, may be all that is needed to control the wheezing. After this treatment the patient often will respond to more conventional therapy. If the patient requires repeated courses of steroids to control attacks, one must consider using them on a long-term basis. However, before beginning such a program of corticosteroid treatment, the patient should be reevaluated thoroughly. Is the diagnosis correct? Is there an infection not being treated in the ears, sinuses, or chest? Is there an important allergen or irritant (e.g., tobacco smoke, or animal dander) still present in the patient's environment?

The recent introduction of beclomethasone (Vanceril®), a potent topical steroid taken by inhalation, has alleviated many of the problems inherent in long-term corticosteroid use, such as adrenal suppression or growth retardation.[6] The usual dose for children (ages six to twelve) is two puffs (100 ugms.) three times a day, with a maximum dose of ten inhalations per day. In adults, the usual dose is two puffs, four times a day, with a maximum of twenty puffs per day. Higher doses will cause adrenal suppression and offer no advantage over systemic steroids. The drug is effective in approximately eighty percent of those treated.

Only a few adverse drug reactions have been reported with the use of beclomethasone. The most common is monilial infection of the pharynx, which occurs in about ten to twenty percent of the patients treated. The usual procedure in this situation is to decrease the dose of beclomethasome and add nystatin (Mycostatin®) orally, until lesions are cleared; the dose of beclomethasone then can be increased. A note of caution must be entertained if the patient has been on prolonged systemic steroids.[7] Adrenal insufficiency can occur as the dosage of oral steroids is decreased when switching to inhaled Vanceril®. One does not know the prolonged effects of local steroids on the respiratory mucosa. In over six years of use in the United Kingdom no problems have been reported. It must be remembered, however, it took many years before the side effects of repeated use of topical steroids on the skin became apparent. There are a few cases of eosinophiilic pneumonia which may be related to beclomethasone therapy.[8]

Beclomethasone may take several weeks to become effective, so most patients will require one or two weeks of oral steroids during that period. Of course, other oral bronchodilator therapy must be continued concomitantly. When the patient has responded well to this treatment program, one then can try to decrease other medications carefully. It is sometimes possible to continue oral bronchodilators and reduce the dose of beclomethasone slowly. Our goal is to reduce the steroids to the lowest dose that will allow the asthmatic patient to enjoy normal activity rather than aiming at complete freedom from asthma. Periodically, an attempt to reduce the dose of corticosteroids, as well as other bronchodilators, should be tried as the patient continues to improve.

Occasionally a patient has a good response to iodide therapy (saturated solution of potassium iodide, Organidin®). These drugs often are helpful during acute episodes, but are not recommended for long-term therapeutic use. Hydration, rest, and adequate diet are all important. The patient should have as much physical activity as he or she can tolerate.

CONCLUSION

There has been a marked improvement over the past few years in the ability of the physician to control the asthmatic patient's wheezing with medications. Prophylactic round-the-clock theophylline, cromolyn, beclomethasone, and newer more specific beta$_2$ sympathomimetics are all advances. However, these drugs do not relieve our responsibility to find, and eliminate, the causes of the asthmatic attacks and to use immunotherapy when indicated. One must not rely on drugs alone. Each patient has his own particular problems and a total regimen must be tailored to the individual, with re-evaluation of this program periodically.

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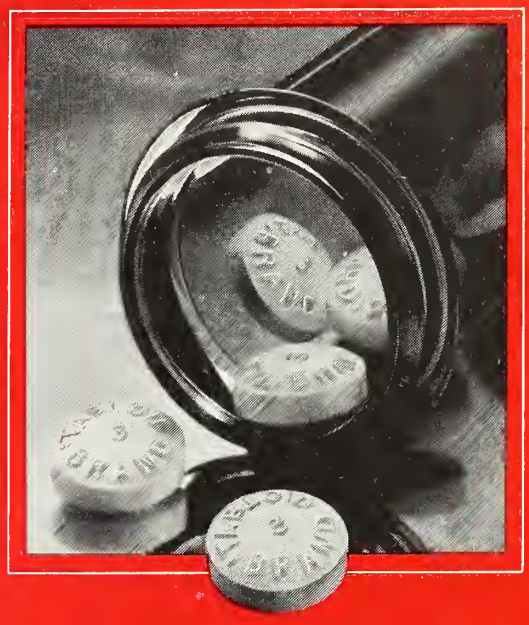
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Neonatal Pneumomediastinum Associated with Renal Anomalies

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Three cases of newborn infants with pneumomediastinum with and without pneumothorax, plus multiple external anomalies, are presented. In all cases these findings were associated with oligohydramnios and severe fetal urologic malformations. The association of oligohydramnios and fetal urologic abnormalities to neonatal respiratory distress and external anomalies is discussed. In cases of unexplained respiratory distress with or without oligohydramnios, pneumomediastinum or pneumothorax must be sought. If found, early examination is recommended to identify fetal urologic anomalies.

The association of renal anomalies with pulmonary hypoplasia and malfunction has been reported by several investigators.[1,2,4,10,11] It is suggested that pulmonary hypoplasia occurs when a fetal renal anomaly gives rise to fetal oliguria and anuria with resultant oligohydramnios. Deformities associated with oligohydramnios include compression of the fetal thorax resulting in pulmonary hypoplasia, deformities of the feet producing club feet, and flattening of the ears and nose known as "Potter's Facies."[10]

In some newborn infants, urologic anomalies are associated with extravasation of air from the pulmonary alveoli into the interstitial tissue and, in severe cases, into the mediastinum and pleural space, manifesting as pneumomediastinum and pneumothorax.[11]

CASE 1

A female infant was born to a 23-year-old Oriental mother (gravida I, para I) at 42 weeks gestation on August 31, 1972. After 21 hours of labor, the infant was delivered by cesarean section due to cephalopelvic disproportion. The Apgar score was four at one minute and seven at five minutes. She was given oxygen but was in moderate respiratory distress when transferred to the nursery. The infant's weight was 5,018 gm and length 51 cm. Oligohydramnios was obvious. The prenatal history was unremarkable. The infant displayed generalized edema with pitting in the extremities. Head was normocephalic with a large occipital caput. The left ear was low set and deformed and the right had poor tone. The ear canals were patent. Respiration was irregular at 60/min. There was minimal intercostal retraction with short apneic spells. Breath sounds were diminished and distant. Heart sounds were regular at 160/min. There was no thrill or murmur. Abdomen was grossly distended. A large mass was palpable in the right side of abdomen extending from the upper to lower quadrant. A similar but smaller mass was palpable in the left side of the abdomen. These masses were firm but had smooth surfaces. External genitalia were normal. Neurological examination revealed hypoactive reflexes.

Chest x-ray disclosed cardiomegaly and the characteristic pattern of pneumomediastinum. The lung fields were compressed superiorly due to marked abdominal distension. There was no evidence of pneumothorax. A supine x-ray of the abdomen revealed an abnormal gas pattern in that a scanty gas pattern was confined to the stomach and proximal bowel. An excretory urogram revealed markedly delayed function in both kidneys. On the two and one-half hour film, the kidneys were relatively well visualized. A poor nephrogram effect was noted. There was a spreading of the calyces

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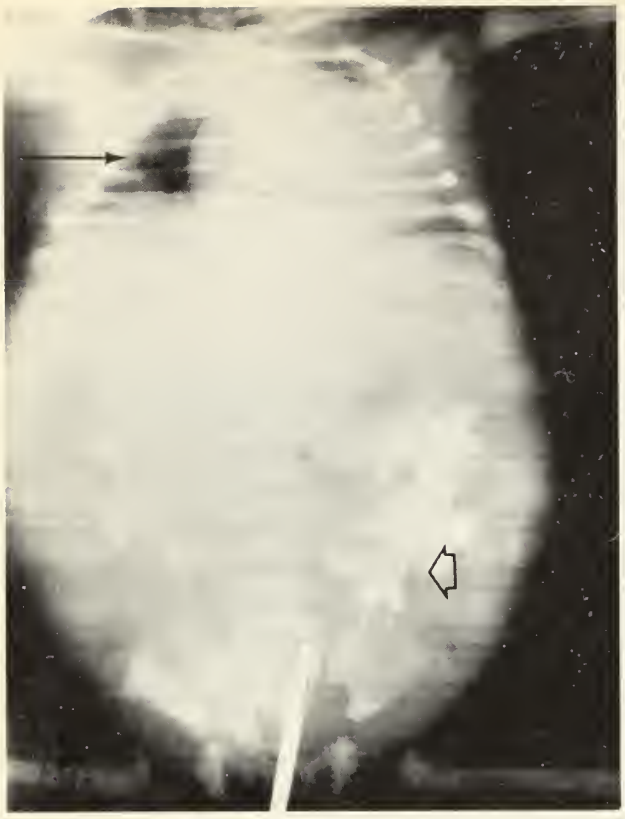


Figure 1: (Case 1) A retrograde pyelogram performed three hours after an excretory urogram reveals spreading and dilation of the left renal collecting system with a large left kidney indicative of infantile polycystic disease. The right kidney reveals similar changes, with contrast remaining from the antecedent excretory urogram. A crescent-shaped hyperlucent air shadow is noted along the right cardiac border indicative of a pneumomediastinum plus cardiomegaly.

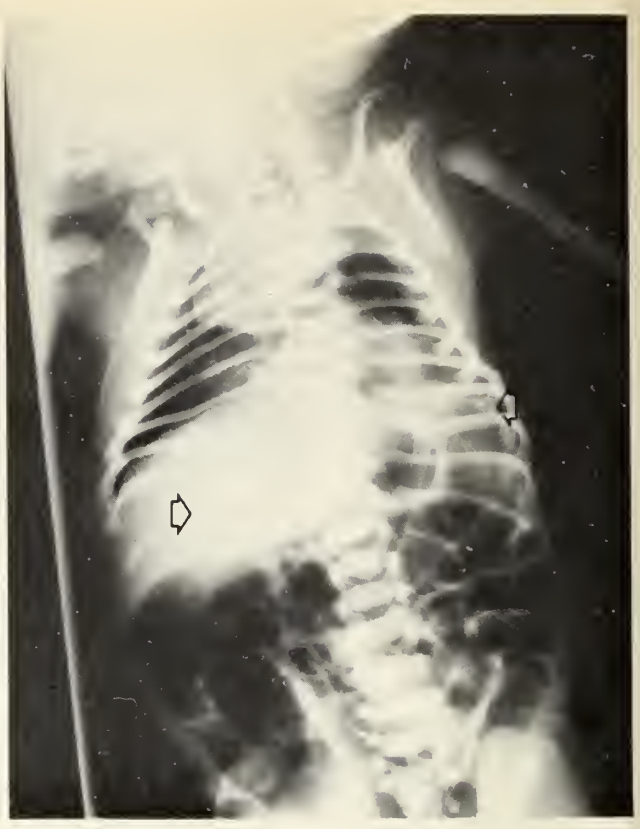


Figure 2: (Case 2) A supine chest radiograph revealing bilateral pneumothorax plus pneumomediastinum with the remaining pulmonary parenchyma displaced superiorly into the lung apices. Dilated small and large bowel loops are noted in the abdomen, indicative of a low colonic mechanical obstruction.

and the collecting systems; the latter appeared somewhat dilated. The ureters and bladder were not visualized. Following excretory urography a cystogram and left retrograde pyelogram (Figure 1) revealed a left renal collecting system with markedly dilated calyces, infundibulum, and pelvis. A right retrograde pyelogram was not performed. These findings were indicative of polycystic disease of the kidneys.

LABORATORY DATA

Hemogram: Hgb 15.7 gm/dl, Hct 48.4%, WBC 17,000, RBC 4.5 million.

Chemistry: BUN was 14 mg/dl, Na 136 mEq/L, potassium 4.8 mEq/L, chloride 98 mEq/L, calcium 8 mg/dl, magnesium 1.4 mEq/L, total bilirubin 2 mg/dl.

Blood gas studies: PaO_2 38 mmHg, $PaCO_2$ 52 mmHg, and P.H. 7.11.

Base excess: (minus 14).

Urinalysis; yellow in color and cloudy, P.H. 5 Albumin 1 + S.G. 1.013—otherwise negative.

Chromosomal study showed a normal 46 xx female karyotype.

A second BUN at 10 hours of life was 17 mg/dl.

The infant's total urinary output was 3.5cc. Her condition deteriorated progressively and she expired at 20 hours of life. A postmortem examination revealed bilateral infantile polycystic kidneys and pulmonary atelectasis. There was no pulmonary hypoplasia.

CASE II

A male infant, a sibling of Case I, was born two years later. It was her second pregnancy and the mother delivered at 38 weeks gestation on October 7, 1974.

The infant was delivered by cesarean section. Marked oligohydramnios was observed. Apgar score was two at one minute and four at five minutes. He did not have spontaneous respiration; he was intubated and transferred to the newborn I.C.U. The infant's weight was 5,500 grams and length was 50 cm. On physical examination the infant had marked generalized edema with pitting in the extremities. The head was normocephalic. The pulse was irregular at 140/minute. There was no cardiac murmur.

The infant was placed on respirator. His abdomen was grossly distended. A large solid horseshoe-shaped mass was palpable in the abdomen. External genitalia were male. Neurological examination revealed marked hypoactive reflexes. Chest x-ray disclosed cardiomegaly and pneumomediastinum. Supine x-ray of the abdomen revealed an abnormal scanty gas pattern and marked abdominal distension.

Laboratory data:

Hgb 13.7 gm/dl	RBC 3.7 million
Hematocrit 44%	WBC 8.3000
Sodium 132 mEq/L	Potassium 6.7 mEq/L
Chloride 90 mEq/L	Co_2 content 19 mEq/L
Calcium 10.3 mg/dl	Po_2 30 mmHg
PCO_2 over 100 mmHg	P.H. below 6.8

Infant was treated with sodium bicarbonate, however, he did not improve and expired at three hours of age.

A postmortem examination revealed bilateral congenital polycystic disease of kidneys and hypoplasia of both lungs associated with intra-aveolar hemorrhages.

CASE III

A premature infant was born to a 19-year-old mother (gravida II para I abortion I) in October, 1973 at 35 weeks of gestation, after a normal delivery. Oligohydramnios and ambiguous genitalia were noted.

Apgar score was two at one minute. Birth weight was 1,800 grams. On physical examination, the infant was cyanotic and had grunting respiration. The head was normocephalic. Ears, nose, and throat were normal. Chest was symmetrical and rales were heard in both lungs. Heart rate was regular at 130/min. There was no murmur. The abdomen was soft. Examination of the umbilical cord revealed a single artery and one vein. There was no meatal opening; neither labian nor scrotum could be identified and the anus was imperforate. The extremities showed bilateral metatarsus abductus, the right foot had only four toes. Neurologically the infant was intact. Chest x-ray revealed pneumomediastinum and pneumothorax. (Figure 2)

Infant expired six hours after birth. A postmortem examination revealed tracheoesophageal fistula, bilateral renal hypoplasia associated with absence of urethra, and an imperforate anus with absence of the rectum.

DISCUSSION

Pneumomediastinum and pneumothorax are among the causes of respiratory distress in the newborn period.[3,6,8,1,2] Possible predisposing conditions include overzealous resuscitation, prematurity, postmaturity, atelectasis, forceful inspiratory efforts, and hyaline membrane disease.

Macklin,[9] in his classic article, aptly has described the pathways for the dissection of air from overdistended alveoli through the interstitium of the lung traveling in the sheaths of the pulmonary blood vessels to form a pneumomediastinum. This can be visualized on chest radiographs before the actual appearance of a pneumomediastinum by pulmonary interstitial edema. Pulmonary interstitial edema actually can press on the pulmonary vessels and interfere with the flow of arterial or venous blood.

Potter,[10] in describing a group of newborn infants with bilateral renal agenesis, reported certain clinical features which they shared. These included widened face, receding chin, prominent fold from the inner canthus to the lower lid, depressed pinnae, and broad hands and clubfeet.

Bain and Scott[1] described a group of infants with fetal oliguria resulting from obstructive uropathy, unilateral renal agenesis with contralateral cystic dysplasia, or bilateral cystic dysplasia, with or without obstruction.

Blanc *et al.*[2] found that all of their cases had one common finding—oligohydramnios. They pointed out that infants with chronic amniotic leakage leading to oligohydramnios had similar facial appearances and abnormalities of the extremities; hypoplastic lungs were found in spite of the presence of normal kidneys. These authors suggested that "Potter's Facies," the clubfeet, broad hands, and hypoplastic lungs associated with renal anomalies and the chronic leakage of amniotic fluid reflected the influence of amniotic fluid and its cushioning effect *in utero*.

Renert[11] *et al.* reported nine cases of newborn infants with pneumothorax and/or pneumomediastinum in whom seven had associated severe fetal urologic anomalies. Oligohydramnios and pulmonary hypoplasia were observed in some cases. Ascites in the newborn, which usually is caused by obstructive urological problems, such as posterior ureteral valves, also can be a factor in pulmonary hypoplasia.

Liberman *et al.* indicated that resuscitation was not the only cause of pneumomediastinum and demonstrated a variety of renal lesions to be the etiologic factor. These authors observed that the presence of one normal kidney even with contralateral renal agenesis was sufficient to prevent the development of "Potter's Facies" and clubfeet. These observations indicate that the associated anomalies are not congenital, but rather secondary to the degree of fetal oliguria.

SUMMARY

Pneumomediastinum in the newborn infant may be associated with renal anomalies and is not necessarily the result of vigorous resuscitation. The three newborns presented in this communication had pneumomediastinum and pneumothorax with severe renal anomalies. It is suggested that associated pulmonary problems are required, and are due in part, to oligohydramnios and the absence of the cushioning effect of amniotic fluid *in utero*. Investigation of the genitourinary tract in newborns with pneumomediastinum and/or pneumothorax is indicated even in the absence of respiratory distress.

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Acute Allergic Alveolitis

STEPHEN A. ANISH, M.D., Wayne

This case report depicts the results of exposure to A. fumigatus in a patient with known allergy to the mold. Diagnosis was made by historical evidence, physical and laboratory findings, and course. Confirmation was made through a positive precipitin reaction using the patient's acute serum. Pathogenesis is discussed relating the immunological basis of the disease.

Although this entity is seen infrequently in our location, exposure to various allergens in industrial, agrarian, and household environments is common.[1-2] Aspergillus is a ubiquitous fungus found in soil and decaying organic matter such as compost heaps, spoiled grain, hay, and rotting wood. Of the many species the most common pathogen in man is *A. fumigatus* (Figure). Less common are *A. niger, A. terreus* and *A. clavatus.*

Three types of pulmonary disorders have been described:

(1) **Colonization** of a pre-existing cavity or fungus ball which may require surgical cure.

(2) **Invasive aspergillosis** occurring in a compromised host. Serum precipitins may be helpful in making the diagnosis. Therapy with amphotericin-B can be begun when the fungus is isolated from blood, tissue, pleural fluid, or other body cavities. Aspergillus in sputum does not necessarily indicate invasive infection by the organism.

(3) **Allergic aspergillosis**—From a clinical standpoint patients with allergic alveolitis rarely spontaneously give their physician a "textbook" recitation of the symptoms. Most patients present with dyspnea, cough, chest tightness, and fever. Fine rales are usually found in the lower lung fields. X-ray demonstrates diffuse pulmonary infiltrates. The presence of serum precipitins to the suspected offending antigen is indirect supporting evidence. Because of nonspecific irritation, skin tests with antigens derived from thermophilic actinomycetes have been shown to be unreliable in diagnosis.

CASE REPORT

A 64-year-old male came to the emergency ward at 1 a.m. complaining of labored breathing, chills, fever, and vomiting. He was in his usual state of health until the evening prior to admission, when he suddenly developed a chill with cough and difficulty catching his breath. He immediately went to bed. His temperature was 40 degrees centigrade orally. The persistent and progressively worsening respiratory distress motivated him to report to the hospital. A careful history revealed that up to 5 p.m. he had been well and working on his daughter's farm. For two hours he had been removing corn from an enclosed bin which was leaky and smelled "moldy." The patient had worked in other grain bins on the same farm but they were not damp and did not smell. Two weeks prior to admission, he had a brief exposure to the same bin and developed some dyspnea which cleared rapidly.

Past history revealed one previous hospitalization for cerebrovascular insufficiency and arrhythmia. He had been maintained on anticoagulants with no recurrences. Fifteen years ago he had relocated from New York City to the Wayne area. His new home was damp and smelled moldy. He had frequent dyspnea and episodes of wheezing. Allergic evaluation demonstrated sensitivity to Aspergillus mold. He moved and did not have any recurrent symptoms. There was no history of chest pain, dyspnea on exertion, edema, hypertension, angina, or myocardial infarction.

Physical examination: B.P. 150/94, p. 120/min. and ir-

regular, R. 30, T. 40°C. rectally. He was plethoric and obese with a few facial cuts. Sclerae were white; pupils were reactive. No neck vein distension was noted and the trachea was in the midline. Scattered fine rales were audible throughout both lower lung fields with an occasional wheeze. No rhonchi were noted. The chest was resonant to percussion. Heart sounds were irregularly irregular at 120 per min. with no murmur, rub, or gallop. Abdomen was soft and no organomegaly was present. Rectum and genitalia were normal. Extremities showed no edema. A small abrasion was found on the left tubia. Neurological examination, skin, and lymph nodes were normal.

Portable chest x-ray revealed cardiomegaly and haziness throughout both lung fields. Repeat chest x-ray 12 hours later showed the heart to be top normal size and the lung fields completely clear. EKG revealed left anterior hemiblock, atrial fibrillation, and a ventricular rate of 120 with nonspecific ST changes probably related to rate. A second EKG done 48 hours later showed atrial fibrillation at a slower rate.

CBC on admission revealed: 8300 WBC's with 88 polys, 11 lymphs, 0 eos.; hemoglobin, 16, hematocrit 48. Blood gases: ph 7.42, Pa $CO_2$23 mmHg., Pa 0_2 73 mmHg. on 0_2 nasal cannula at 3 L/min. BUN 14 mg/dl. Serum electrolytes Na 132 mEq/L, K4 mEq/L, Cl109 mEq/L, CO_2 21 mEq/L. Prothrombin time prolonged because of Coumadin® therapy.

COURSE

The patient initially was treated with oxygen at 5L/min., hydrocortisone 100 mg. I.V. immediately and every six hours. Blood cultures, and sputum and urine cultures were obtained and none showed bacterial growth. Sputum for gram stain failed to demonstrate a predominant bacterial type; few WBC's were seen. Digoxin 0.5 mg. was administered initially intravenously and later titrated according to his rate. No urinary diuresis occurred. Daily prothombin times were done to regulate his Coumadin® dose.

Follow-up chest x-ray showed complete clearing of the diffuse alveolar infiltrates in 12 hours. Blood gas studies 18 hours later on room air were ph 7.42, Pa CO_2 36 mmHg., Pa O_2 98 mmHg. He was moved from ICU to the regular floor within 24 hours. Blood was obtained for serologic studies and demonstrated A. fumigatus precipitins on Ouchterlony double diffusion in agarose. No further dyspnea occurred. He defervesced within six hours and had no recurrent fever despite discontinuance of steroids in 24 hours. He was discharged two days later after full ambulatory status was obtained.

DISCUSSION

A. fumigatus spore inhalation can result in aspergilloma formation in pulmonary cavities or cysts, invasive aspergillous in immunodeficient patients, allergic bronchopulmonary aspergillosis in atopic patients, and extrinsic allergic alveolitis in nonatopic patients.[3-6] The patient described had neither cavity, cyst, nor atopy. There was no evidence of invasive aspergillosis on chest x-ray. He did have a pulmonary hypersensitivity reaction due to the inhalation of A. fumigatus spores as supported by the history of allergy, two known reactions, one requiring this hospitalization, and serum precipitins to A. fumigatus. The A. fumigatus spore measures 2.5 to 5 mm. in diameter and may be deposited in peripheral lung tissue through inhalation. Similar findings have been described in malt worker's lungs due to inhalation



Figure—*Aspergillus Fumigatus.* Typical condiophore x 820. Line drawing from Conant *et al. Manual of Clinical Mycology*, second edition. Philadelphia, Saunders.

of A. clavatus.[7,8]

The pathogenesis of this patient's disease could have been mediated through an intrapulmonary Arthus-type reaction to the inhaled antigen.[9] Sensitization occurs to A. fumigatus which grows in moldy corn. Reactions occur within a few hours after exposure. The reaction results in both systemic and pulmonary manifestations. The pulmonary reaction has been considered as an example of Type III (immune complex mediated) immune response according to the classification of Gel and Coombs. However some patients exhibit an immediate wheezing syndrome more typical of Type I (Ig.E. or reagin mediated) immune response. Further, the histological findings in the lung in allergic alveolitis are typically those more characteristic of Type IV (cell mediated) hypersensitivity with round cell infiltration of a granulomatous nature. In fact, evidence is now accumulating that both cellular and humoral immune mechanisms may be operative and the relative importance of each in the pathogenesis of these pneumonitities may depend on the chronicity of exposure, the nature of the antigen and probably other factors as well.[10] If exposure is heavy (as in this case) the patient will exhibit shaking chills and temperature elevation from 39 C. to 40.5 C. There will be profuse sweating and malaise. Generalized joint aches can occur. This is followed by a nonproductive cough and shortness of breath on slight exertion. The symptoms usually last for at least 12 to 16 hours and the patient recovers provided repeated exposure is eliminated. Physical examination usually reveals chills, tachypnea, fever, bibasilar rales, and tachycardia. Laboratory tests show slight to moderate elevation of neutrophils with a slight left shift. If eosinophilia is present, it does not exceed 10 percent. Roentgenographic examination at the time of acute exacerbation demonstrates coarsening of the bronchovescicular markings, nodulations, and confluent

areas of reticulation throughout both lung fields. Pulmonary function shows mild impairment which reverses rapidly with recovery. The diffusing capacity is decreased as is the compliance. If repeated episodes occur, compliance remains low. Arterial oxygen concentration is decreased and may reach 50 mmHg. Pa CO_2 is low or normal. Blood gases return to normal rapidly. The disease occurs in subacute and chronic stages related to the frequency and duration of exposure. Treatment requires removal of the offending antigen and use of corticosteroids when complicating circumstances are present or after prolonged exposure. Unfortunately, however, particularly in the more chronic exposures, there is a variable amount of irreversible change and corticosteroid therapy may be of limited benefit.

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Giant Hyper-Secreting Villous Adenoma of the Rectum

I. SALAHI, M.D., W. TEVLIN, M.D., L. JAWNY, M.D.,
R. GIOBBE, M.D., G. YELIN, M.D., Kearny

Abdomino-sacral resection was used for removal of a giant hyper-secreting villous adenoma of the rectosigmoid colon. The tumor was associated with severe fluid and electrolyte depletion.

Villous tumors of the large intestine first were studied adequately in 1948 by Sunderland and Binkley.[1] Hyper-secreting villous adenoma was recognized and reported by McKittrick and Wheelock in 1954.[2] This spectacular syndrome of hyper-secretion is seen only occasionally in patients with giant villous adenoma. The profound hypokalemia, hyponatremia, hypochloremia, and prerenal azotemia, which are associated with these lesions, have been described in many excellent reviews. This report adds another instance of this distinctive neoplasm which is unusual because of its size. The tumor measured 16 cm in maximum diameter which represents the largest villous adenoma recorded in the literature. In addition, the method of surgical management, namely resection of the rectum with preservation of the anus, is an uncommon procedure for this lesion.

CASE REPORT

A 58-year-old male was admitted to West Hudson Hospital because of increasing weakness and watery diarrhea for the past few months. This was associated with a 10 to 20 pound weight loss. He had been in good health, but did recall occasional bouts of diarrhea not associated with nausea, vomiting, or abdominal pain. The patient appeared to be moderately dehydrated, but not chronically ill. Blood pressure was 120/80 mmHg. There were no significant abnormalities on physical examination. On rectal examination, a soft tumor was felt on the posterior rectal wall. The upper margin of the tumor could not be reached with the examining finger.

Laboratory findings on admission showed a white blood count of 16,300 with 81 polys and 17 lymphs. Hemoglobin was 20.4 gm and hematocrit 57.3%. Blood chemistry revealed a level of potassium of only 2 mEq per liter; sodium was 126 mEq and chlorides were 61 mEq. Blood urea nitrogen was elevated to 120 mg/dl and creatinine to 5.2 mg/dl.

Vigorous fluid therapy was initiated. Guided by central venous pressure which was determined frequently, the patient received 200 mEq of KCl daily until hypokalemia was corrected. After correction of the electrolyte imbalance, a barium enema x-ray examination was performed; this disclosed a large rectal tumor. (Figures 1, 2)

Proctoscopy showed a large polypoid obstructing tumor at about six cm from the anus. Pathological examination of multiple biopsies of the tumor revealed the lesion to be a histologically benign villous adenoma. At surgery a large soft tumor extensively filled the upper and middle thirds of the rectum. (Figures 3, 4) The tumor was resected, using the adbomino-sacral procedure, with restoration of continuity.[3] Histologic examination (Figure 5) showed a predominantly benign pattern with regular arrangement of epithelial cells. However, a few isolated foci of cell atypism with rare mitotic figures were seen. (Figure 6) Multiple sections showed no

*From the surgical and medical departments, West Hudson Hospital, Kearny, New Jersey.





Figures 1 and 2—Radiographic examination revealed a very large, bulky lesion of the rectum about 16 cm in length, encircling the entire lumen and causing an extensive filling defect. The margins of the tumor are rough, revealing deep clefts, and the surface of the lesion had a "cobblestone" appearance. These findings are characteristic of a villous adenoma.



Figure 3—Overall view of the villous adenoma. Note sharp margin of separation of neoplasm from adjoining normal mucosa.



Figure 4—Close-up view of the neoplasm to demonstrate the villous (papillary) pattern.

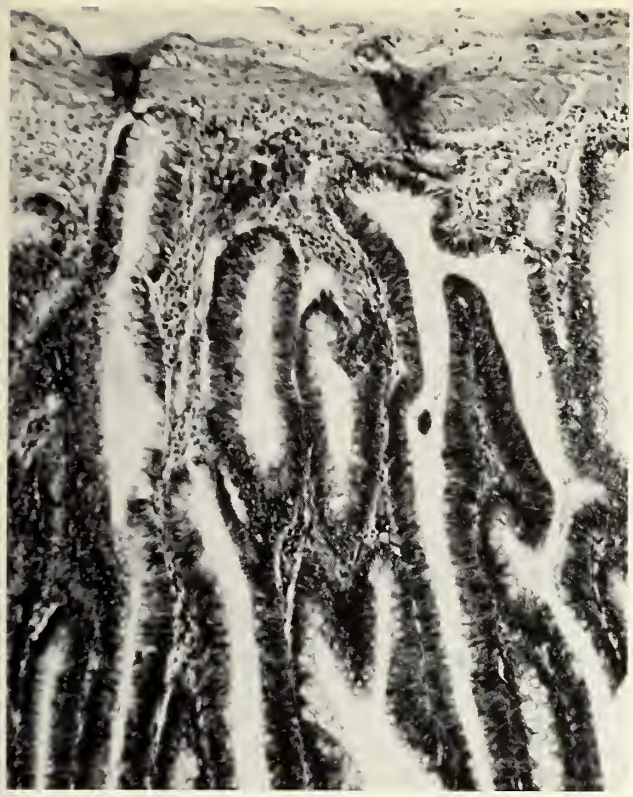


Figure 5—Note the regular, though crowded, pattern of single layers of epithelial cells along the delicate villous processes (x 50).

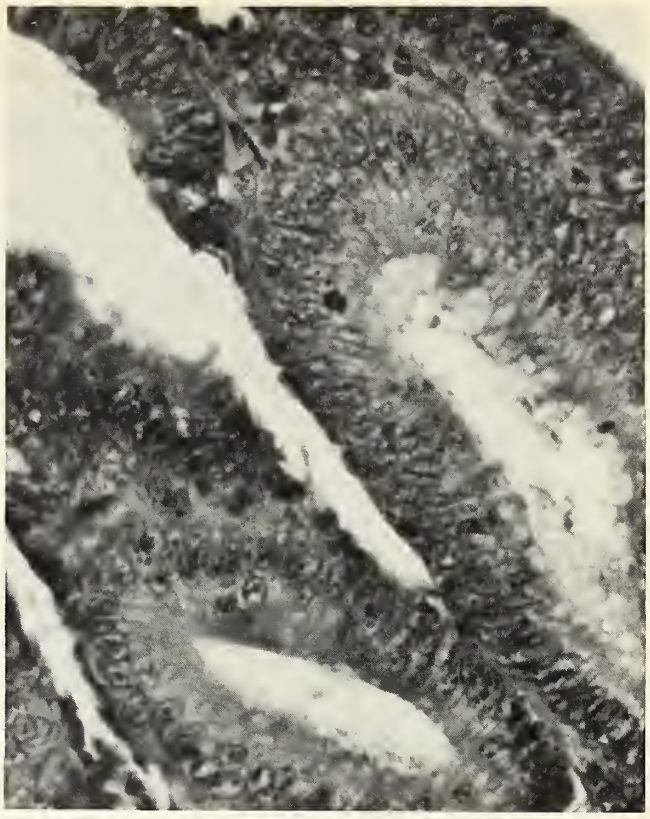


Figure 6—Area of moderate cell atypism with uneven location of nuclei (loss of polarity), however, there is no progression beyond the basement membrane (x 100).





Figures 7 and 8—Post resection radiographs revealed a well-functioning anastomosis with some extravasation of barium about the slight surgical defect.

evidence of infiltration or other stigmata of malignant change.

The post-operative course was complicated by prolonged ileus and partial small bowel obstruction, which necessitated nutritional support with intravenous hyperalimentation and small bowel decompression with a long Anderson tube. After adequate small bowel decompression, a barium study through the tube revealed no small bowel obstruction, but there was evidence of fecal fistula. (Figures 7, 8) For this reason, a transverse colostomy was made. The patient's condition improved satisfactorily and he was discharged from the hospital. The colostomy was closed two months later. Anal function was restored with satisfactory control of the sphincter.

DISCUSSION

The severe fluid and electrolyte loss associated with giant hyper-secreting villous adenoma is potentially dangerous. A few deaths have been reported in the literature, where the diagnosis was missed and villous adenoma was found at autopsy. However, this bizarre pattern is not the usual one.

Villous adenoma is very soft and velvety in nature and is easily missed on rectal examination. Consequently, repeated sigmoidoscopy may be needed before the tumor is finally discovered. Pseudodiarrhea, usually worse in the morning, is pathognomonic of this tumor.

The abdomino-sacral procedure, with preservation of the anus, for resection of a lesion in the middle third of the rectum is very much appreciated by the patient since it obviates the need for a permanent colostomy. The most common potential complication of this surgical approach is the development of a fecal fistula. Nevertheless, patient acceptance of this possible risk outweighs the necessity of a colostomy as part of the more conventional technique for low and mid-rectal tumors.

SUMMARY

To our knowledge, this case of giant hyper-secreting villous adenoma of the rectum represents the largest benign villous adenoma ever reported. The choice of surgical management of this tumor was not the conventional abdomino-perineal resection, but rather abdomino-sacral resection with preservation of the anus with excellent continence.

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Characterization of the Malignant Ventricular Premature Impulse in Acute Myocardial Infarction*

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This review article critically examines recent and past experimental and clinical information pertaining to the malignant potential of VPIs in patients with AMI. It seems reasonably clear that the electrophysiologic status of the experimentally infarcted heart is more important in the genesis of serious VAs than are the specific features of VPIs, and that VPIs, VT and VF are varying manifestations of these electrical dysfunctions. Prophylactic anti-arrhythmic therapy may be indicated in all patients with proved or suspected AMI.

While ventricular premature impulses (VPIs) occur in nearly all patients with acute myocardial infarction (AMI), most VPIs are benign, but some are associated with more serious ventricular arrhythmias (VAs) and often are referred to as "malignant" VPIs. Because of obvious prognostic and therapeutic implications, years of careful clinical observation and rigorous laboratory investigation have been devoted to identification of this arrhythmogenic potential; yet, the precise characteristics of the malignant VPI in the setting of AMI remain undefined.

Historically, the original investigators seemed preoccupied with the concept that the degree of prematurity of a given VPI determined its malignant potential. In 1940, Wiggers defined a "vulnerable period" in the canine ventricle during which single electrical shocks elicited repetitive ventricular firing.[1] Some 20 years later, Smirk and Palmer made similar observations in man and described the "R-on-T" phenomenon, i.e., the production of ventricular arrhythmias by VPIs whose R wave interrupted the T wave of the previous normal impulse.[2] With the advent of coronary care units (CCUs) and constant monitoring devices, R-on-T was widely accepted as the prime marker of the malignant VPI. More recent experimental and clinical evidence strongly suggests that this arrhythmogenic tendency is not restricted to "early" VPIs, i.e., those exhibiting the R-on-T pattern, and that ventricular fibrillation (VF) and ventricular tachycardia (VT) may be initiated by VPIs and even atrial premature impulses (APIs) occurring later in diastole.

In addition to degree of prematurity, other features of VPIs have been assigned to malignant or benign categories, and these are listed in the Table. The purpose of this review is to examine critically some recent experimental and clinical information pertaining to the problem, and to attempt to synthesize contemporary ideas about the electrogenesis and prevention of VAs in AMI.

EXPERIMENTAL DATA

In 1963, Lown, using relatively large, direct current stimuli applied to the chest wall of normal, intact animals, confirmed the earlier studies of Wiggers.[3] VF never occurred if the stimuli were applied outside the vulnerable period, an interval of 30 msec. immediately preceding the apex of the T wave. This observation remained unchallenged until Carroll and his associates evaluated spontaneous VT and VF in dogs with AMI due to coronary artery ligation (CAL).[4] In their experiments, T wave interruption (R-on-T) invariably preceded fatal VF, but 76 percent of episodes of VT in survivors were initiated by VPIs that appeared after the T wave.

In a series of papers published between 1969 and 1972, Han showed that experimental AMI lowered the VF threshold and made the ventricle more susceptible to VT and VF because of localized areas of depressed excitability and

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Table

Benign VPIs		Malignant VPIs
Late	Degree of Prematurity	Early (R-on-T)
Right Ventricle	Site of Origin	Left Ventricle
Re-entry	Electragenesis	Parasystole
Absent	Interpolation	Present
Intact	V-A Conductian	V-A Black
<30/Hr.	Frequency	>30/Hr.
Absent	Bursts or Salvas	Present
Absent	Multifarmity	Present
<140 Msec. (Septal)	QRS Duratian	>140 Msec. (Parietal)

conductivity that favored re-entrant activity, fractionation of excitation and total disorganization of impulse propagation.[5-7] While ineffective in the normal heart, VPIs with long coupling intervals (CIs) precipitated VF regularly in dogs with AMI due to CAL, presumably because propagation of the VPI was delayed through the ischemic area allowing for first localized and then generalized re-entry with eventual disorganization to VF. He also indicated that these effects were accentuated during bradycardia, owing to increased temporal dispersion of repolarization that predisposed the ventricle to re-entrant activity and subsequent VF.

The work of Epstein and his co-investigators in 1973 provided somewhat different data.[8] They studied 72 closed-chest, conscious dogs with AMI in whom VPIs or VT developed spontaneously in 52. VF occurred in 18 and was always preceded by a VPI whose CI was less than 0.43 sec. VF was not seen with VPIs whose CI was greater than 0.43 sec. Accordingly, VPIs or VT with CIs less than 0.43 sec. were considered benign. In contradistinction to Han's results, correction of bradycardia by atrial pacing or atropine suppressed only the benign VAs and failed to prevent VF. Furthermore, lidocaine suppressed benign VAs in three of five dogs but failed in all six dogs with malignant VAs. Combining atropine and lidocaine offered no advantage over either drug alone. These authors concluded that, in the early phases of experimental AMI, the malignant potential of VPIs could be defined by their CIs, and that while increasing heart rate or infusing lidocaine effectively abolished benign VAs, these interventions were considerably less effective in preventing VF.

Waldo and Kaiser used bipolar epicardial electrograms to show continuous electrical activity or "localized fibrillation" within the infarcted myocardium of dogs subjected to CAL.[9] When localized fibrillation persisted beyond the T wave of the preceding impulse, it was associated with VPIs; sustained, localized electrical activity led to generalized VT and VF. The occurrence of localized fibrillation was favored by the electrophysiologic properties of infarcted myocardium, i.e., enhanced threshold of excitability, shortened refractory period, decreased conduction velocity and increased temporal dispersion of recovery. The implication was that localized fibrillation spread to the rest of the ventricles, perhaps related to the mass of infarcted tissue. The gist of this important paper was that the genesis of serious VAs was related more to the electrophysiologic status of the infarcted myocardium than to the specific characteristics of preceding VPIs.

Williams and his associates also employed bipolar epicardial electrograms to evaluate malignant VAs in open-chest dogs with AMI due to CAL.[10] They showed delay and fragmentation of potentials recorded from the ischemic area resulting in prolongation of the ventricular activation time (VAT). Induced VPIs productive of ventricular fibrillation were associated with progressive increases in VAT, while VAT progressively decreased in self-terminating ventricular tachycardia not associated with VF. Serious ventricular arrhythmias always followed those impulses that demonstrated the greatest delay of the ischemic zone potential and increased VAT; they were in no way related to the coupling interval of VPIs. It was felt that the electrophysiologic alterations caused by acute myocardial infarction markedly increased the duration of the ventricular vulnerable period so that either early or late VPIs could cause VT or VF. This group suggested that in AMI, the malignancy of a VPI was determined by the underlying alterations of electrical activation and was not related to its degree of prematurity. Since it was not possible to measure regional activation time except by direct electrode recordings, they deemed it ". . . prudent to ascribe potential malignant properties to all VPIs, irrespective of their diastolic timing, during the early state of AMI."

Experiments by El-Sherif confirmed these data and demonstrated fragmentation and delay of local electrograms resulting in dispersion of total VAT and a close temporal relationship between delayed activation and the onset of early phase VAs.[11] These studies also suggested that the electrophysiologic mechanism for VAs was an irregular conduction disorder of ischemic myocardium, specifically of the Wenckebach pattern. Interestingly, the conduction disorder was tachycardia-dependent, and slowing of the heart rate resulted in improved conduction and coincident disappearance of VAs. This protective effect of bradycardia was quite different from that previously reported by Han and others.[5-7] Vagal-induced slowing of the atrial rate often abolished the VAs, suggesting that, in the presence of marked dispersion of ventricular activation, rapid atrial rates could induce VPIs, multiple re-entrant circuits, and subsequent VF. This provided a rational explanation for subsequent clinical observations of VAs initiated by atrial premature impulse. Most of the malignant VAs in this study were begun by late, coupled VPIs with no evidence of the R-on-T phenomenon. He postulated that the ventricular vulnerable phase could occupy most of the cardiac cycle in AMI because of marked dispersion of ventricular activation so that the malignant potential of VPIs could not be defined solely by their degree of prematurity. This study also showed, for the first time, that delayed, asynchronous activation could be detected by intracardiac electrode catheters as well as by direct epicardial or intramyocardial leads. He concluded that VPIs were not the primary culprit in the production of VF, but rather that they were secondary manifestations of a more basic problem related to the degree of dispersion of ventricular activation.

Wit also de-emphasized the role of VPIs in the genesis of VAs in experimental AMI, relating them more to relatively new concepts concerning the altered electrophysiology of infarcted myocardium.[12] He and others showed that the transmembrane electrical activity responsible for early phase VAs resulted from different ionic mechanisms than those of non-ischemic cells. Depolarization of normal ventricular cells occurs rapidly owing to a large influx of sodium ions via a strong inward current through specific membrane channels. A second, slower and weaker inward or depolarizing current occurs later mediated by calcium ion influx through "slow" membrane channels. In ischemic cells there is a reduction in resting membrane potential causing inactivation of rapid sodium-dependent depolarization, but having no

effect on slow channel depolarization. This "slow response" favors the appearance of VAs because of its association with slow conduction, unidirectional block and asynchronous or fragmented activity. These phenomena predispose to the development of re-entrant circuits resulting in single or multiple VPIs, VT, or VF. Here again, the experimental data suggested that VPIs were not causally related to VT and VF, but rather that all VAs were engendered by fundamental electrophysiologic alterations in infarcted myocardium.

CLINICAL OBSERVATIONS

As stated previously, early observations in CCUs indicated that most patients with AMI had VPIs, and many investigators believed that ventricular ectopic activity, of whatever degree, was a harbinger of VF. Since the consensus was that early mortality from VF could be accurately predicted by the detection of VPIs, aggressive anti-arrhythmic therapy with lidocaine and other agents was urged, and the general philosophy of therapy in AMI shifted from resuscitation to prevention. In Julian's series, VPIs were detected in 67 of 100 consecutive patients with AMI studied prospectively with an oscilloscopic monitoring device.[13] In 32 patients with frequent VPIs (more than one per 10 normal beats), VF occurred in six, while only one instance of VF was seen in 35 patients with less frequent VPIs. Four of seven patients with early (R-on-T) VPIs died of VF, while two of 10 with bursts of VPIs developed VF, and two of six with multifocal VPIs had this lethal arrhythmia.

In a series of papers published between 1967 and 1971, Lown evaluated 600 patients with AMI and found only one instance of VF without preceding VPIs.[14-16] VPIs occurred in 80 percent, and especially ominous features included frequency, bursts or salvos, multiformity and marked prematurity. Based on these observations, he developed a grading system for VPIs:

Grade 0—no VPIs
" 1—occasional VPIs
" 2—frequent VPIs (more than 30/hr.)
" 3—multiform VPIs
" 4A—VPIs in couplets
" 4B—VPIs in salvos
" 5—early VPIs (QR/QT less than 1.0)

Higher grades implied electrical instability and augured sudden death from VF. Lown also reported that lidocaine was effective in abolishing 88 percent of VAs with ". . . nearly complete prevention of VF."

At approximately the same time, Pantridge and Adgey described their original findings in patients with AMI employing a mobile CCU ambulance that allowed them to provide pre-hospital care.[17] They noted a remarkable incidence of bradyarrhythmia in patients seen within the first hour of onset of AMI and felt that slow heart rates predisposed to VF based on experimental data that indicated a lowering of VF threshold during bradycardia.[5-7] Since the majority of early deaths were the result of VF, these authors advocated prophylactic administration of atropine, a measure that was universally accepted.

In 1968, Lawrie's group published their findings in 600 patients with AMI of whom 8.3 percent developed VF.[18] Their report included some controversial and provocative features. "Warning" VPIs occurred rarely and, even when detected, they were not present long enough to allow for institution of prophylactic therapy. Of 12 patients who developed VF and were monitored for more than 15 minutes, only two had VPIs that could have been treated. Furthermore, in this series, sinus bradycardia seldom preceded VF. These authors also argued that lidocaine had not been totally effective in preventing VF since there was only a slight reduction in the incidence of VF after adopting Lown's regimen for lidocaine prophylaxis.

Brown and his associates noted primary VF in 20 of 851 patients with proved AMI.[19] Five had no preceding VPIs, three had infrequent VPIs and the remaining 12 had either VT or frequent VPIs. Eight developed VF while receiving lidocaine and/or procainamide. None of the patients showed bradycardia prior to VF. These investigators reinforced Lawrie's impression that the increasing use of anti-arrhythmic drugs had effected no significant reduction in the incidence of primary VF.

Moss reported on ECG tape recordings in 33 patients with AMI obtained in satellite, industrial CCUs.[20] This pre-hospital study disclosed 12 patients who had either VT or VF, all of which were preceded by early VPIs (QR/QT less than 1.0). In contrast to the pre-hospital observations of Pantridge and Adgey,[17] sustained sinus bradycardia and VPI frequency were not related to serious VAs.

In his review of tachyarrhythmias in AMI, De Sanctis confirmed the frequency of VPIs and indicated that VAs could be initiated by VPIs ". . . outside the vulnerable phase"; his figure ten is an excellent example of VT precipitated by a barely premature impulse.[21] He also supported Salazar's impression that parasystolic VAs in AMI had a benign prognosis; prior to these publications, it generally was agreed that parasystolic VAs had a more dire prognosis than re-entry mechanisms.[22] His review of the literature disclosed that while prophylactic anti-arrhythmic therapy reduced VPIs, overall mortality was generally similar in treated and control groups.

Wyman and Hammersmith studied 1165 consecutive patients with AMI and found 12 instances of primary VF.[23] Seven of these patients had very rare or no VPIs preceding the VF. Their review of the literature revealed that 25 to 83 percent of patients with primary VF had no "warning arrhythmia." Accordingly, they advocated prophylactic lidocaine for all patients with suspected AMI to be started in the emergency room prior to CCU admission. In cases where VPIs were not controlled with lidocaine, intravenous procainamide was instituted. Employing this regimen, their prevalence rate of primary VF dropped from 6.5 percent (9 of 139 cases) to 0.3 percent (3 of 1026 cases).

In 1974, De Soyza and his associates, employing 24-hour Holter tape recordings, studied the role of the coupling interval of VPIs in the production of VT in patients with AMI.[24] They evaluated 52 consecutive patients with AMI in whom Holter monitoring was begun within 24 hours of the onset of chest pain. In 27 patients, 131 episodes of VT were documented. The mean CI of VPIs initiating the runs of VT was not significantly different from the mean CI of VPIs not initiating VT in the 27 patients who had VT, nor did it differ from the mean CI of VPIs in the 25 patients without VT. Only 12 percent of VPIs causing VT had a prematurity index less than one, while VPIs with long CIs frequently precipitated serious VTs. The mean heart rate preceding VT was not significantly different from that recorded in the 25 patients without VT, and only 15 percent of the runs of VT occurred during sinus bradycardia. Interestingly, frequency of VPIs and the occurrence of accelerated idioventricular rhythm (AIVR) were of importance. [25] In the 27 patients with, the median number of VPIs was five per 1000 normal

beats, while the median number of VPIs in the 25 patients without VT was only 0.7 per 1000 normal beats (P0.005). Accelerated idioventricular rhythm occurred in 59 percent of the 27 with VT, while only 8 percent of those without VT had AIVR (P0.005). This group concluded that the degree of prematurity of a VPI was a poor predictor of its malignancy, but that increased frequency of VPIs and AIVR were associated with an increased incidence of ventricular tachycardia.

Lie and his group also used continuous tape recordings to evaluate the events preceding primary VF in patients with AMI.[26] They studied 262 consecutive patients within six hours of the onset of chest pain and detected primary VF in 20 (7.5 percent). Only 12 patients had "warning arrhythmias" and only two had preceding bradycardia. Early VPIs (QR/QT0.85) initiated nine episodes of VF, while 11 were begun by late VPIs (QR/QT0.85). VPIs with a left bundle branch block configuration caused six episodes of VF, while six were begun by VPIs with a right bundle branch block pattern, and eight were indeterminate. These authors concluded that "warning arrhythmias" were not helpful in predicting VF and that there was no clear relationship between bradycardia and VF in man. They also stated that the site of origin of a VPI, as indicated by QRS contour, was not a reliable determinant of malignancy, a finding contrary to previous information which indicated that "left ventricular" VPIs had a more ominous prognosis.[27] Even more disturbing is a recent study by Josephson and his colleagues[28] indicating that the original criteria of Rosenbaum[29] for localizing the origin of VPIs may be erroneous. Finally, Lie's paper strengthened the argument that the degree of prematurity of a VPI was a poor predictor of its malignancy and that slow fragmented activation of ischemic myocardium was a major determinant of malignant VAs. "Our data in man indicate that the malignancy of a VPI is not only related to its CI."[26]

El-Sherif detected primary VF in 20 of 450 consecutive patients with AMI admitted within 24 hours of onset of their chest pain.[30] Interestingly, eight patients were already receiving lidocaine when VF occurred. "Warning arrhythmias" preceded VF in 11 patients (58 percent), but these also were present in 236 of 430 patients (55 percent) without VF. In seven patients, VF was begun by late VPIs (QR/QT1.0), while 10 were initiated by early VPIs (QR/QT1.0). However, early VPIs also occurred in 49 percent of patients without VF. In one case, an API with aberrant intraventricular conduction produced VF. He stated that in AMI, the duration of the vulnerable period was prolonged, and ". . . that the arrhythmogenic potential in AMI is not confined to those VPIs showing the R-on-T phenomenon."[30]

Recently, Bigger critically reviewed the Lown grading system for VPIs and emphasized its limitations and shortcomings.[31]. The majority of his patients in grades 1 and 2, because of VPI frequency, actually were eligible for higher grades because of characteristics of their VAs other than frequency. Another problem in this group of 100 patients with AMI was the appearance of important differences between peak hourly VPI counts, as advocated by Lown for determining frequency, and overall average VPI frequency. Theoretically, a patient with 30 VPIs per 24 hours could be in grade 2 if all of his VPIs occurred in one hour, while another patient with 696 VPIs per 24 hours could be placed in grade 1 if he had exactly 29 VPIs per hour for 24 hours! Finally, Bigger argued that there was no proof that higher grades indicated higher risks of subsequent adverse outcome, especially with respect to the R-on-T phenomenon.

After reviewing the literature on the R-on-T phenomenon, Engel, Meister, and Frankl remarked that "R-on-T does not appear to be the critical determinant of VT or VF in AMI."[32] Because of apparent inability always to identify the harbinger of VAs, they defended the argument for prophylactic anti-arrhythmic therapy in patients with AMI. I strongly recommend careful perusal of this comprehensive "update and critical review."

Finally, Roberts and his associates reported their experience with VAs in 38 patients with AMI studied with magnetic tape recordings that were analyzed by an Argus/H computer system.[33] All of the patients had VPIs and 29 had a total of 337 episodes of VT. "Late" VPIs (QR/QT>1.0) initiated 78 percent of the runs of VT, while only 22 percent were begun by "early" VPIs (QR/QT<1.0). Only two episodes of ventricular fibrillation were detected, and both were initiated by late VPIs. These authors concluded that ". . . the frequent initiation of repetitive ventricular depolarizations by late premature ventricular complexes attests to their potential importance as warning arrhythmias on the one hand and as precipitants of malignant arrhythmias that may require pharmacologic suppression on the other."

Our own experience with harbingers of ventricular tachycardia in AMI was recently published, and it tends to support contemporary experimental and clinical concepts.[34] We detected 68 instances of VT in 42 patients with AMI confirmed by clinical, electrocardiographic, and enzymatic abnormalities. Frequent VPIs (6/min.) were associated with 45 episodes of VT, while multiform VPIs were seen in 11. "Right ventricular" VPIs initiated 24 runs of VT, while "left ventricular" VPIs were responsible for 39; this difference was not significant. APIs were responsible for five instances of VT, a phenomenon referred to previously.[11,30] Only 17 episodes of VT were related to early VPIs (QR/QT<1.0), while 46 were begun by late VPIs (QR/QT>1.0). The mean duration of VT related to early VPIs was 22 impulses, while VT begun by late VPIs or APIs was significantly shorter, eight and six impulses respectively. Lidocaine abolished only four runs of VT due to early VPIs, but was much more effective in VT due to late VPIs and APIs; it terminated 41 bouts of VT due to late VPIs and all five instances of VT begun by APIs. Interestingly, six episodes of VT due to early VPIs deteriorated to VF despite Lidocaine, but VF never ocurred when VT was begun by late VPIs or APIs.

EPILOGUE

While most patients with AMI have VPIs, relatively few have more serious VAs. The consensus of recent experimental and clinical information is that these are not causally related, but rather that VPIs, VT and VF are all manifestations of basic electrophysiologic dysfunctions of infarcted myocardium.

Critical review of the experimental data yields the following impressions:

1. The experimentally infarcted ventricle has a lower VF threshold and a longer vulnerable period than the normal ventricle.

2. Spontaneous VPIs affect the ventricle differently from those produced by artificial methods such as direct current shocks or pacemaker stimuli.

3. Heart rate and degree of prematurity of VPIs are not exclusive determinants of malignancy.

4. The electrophysiologic status of the experimentally infarcted heart is more important in the genesis of VAs than

the specific features of VPIs. The underlying electrical alterations include localized VF, prolonged VAT, abnormal dispersion of ventricular activation and slow channel depolarization.

Analysis of recent clinical observations provides these thoughts:

1. The Lown grading system is not sacrosanct; there are numerous pitfalls and limitations.

2. While bradycardia may be a harbinger of VF very early in the course of AMI, the significance of its later occurrence is not at all clear.

3. Neither degree of prematurity nor site of origin is a precise determinant of malignancy of a given VPI.

4. VF can occur without preceding VAs and may even be initiated by APIs; this intriguing relationship between APIs and VAs demands further experimental and clinical investigation.

5. Since the basic electrophysiologic faults in AMI, such as abnormal dispersion of ventricular activation, are not identifiable by current clinical methods, and since the VAs that occur in AMI are related more to these problems than to specific features of VPIs, I believe that prophylactic antiarrhythmic therapy is indicated for all patients with proved or suspected AMI. On the other hand, such therapy is no guarantee that fatal VF will not occur. Caveat emptor!

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List of abbreviations
(in alphabetical order)

AIVR	—	accelerated idioventricular rhythm
AMI	—	acute myocardial infarction
API	—	atrial premature impulse
CAL	—	coronary artery ligation
CCU	—	coronary care unit
CI	—	coupling intervals
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Cirrhotic Patient Hemorrhaging From Gastroesophageal Varices: Role of Selective Portal Venous Decompression

FREDERICK A. REICHLE, M.D., Philadelphia

Hemorrhage secondary to hepatic cirrhosis with portal hypertension and gastroesophageal varices should be controlled promptly by balloon tamponade and adjunctive non-operative methods. Once hemorrhage has been controlled, elective portal venous reconstruction must be considered to prevent recurrent hemorrhage with its associated mortality. The expected survival has improved because of newer, selective portasystemic shunts (distal splenorenal, mesocaval).

Although hepatic cirrhosis with portal hypertension is caused by parasites (schistosomiasis) in more than 90 percent of cases as a world-wide problem, quite the reverse is true in the United States where 90 percent of portal hypertension and hepatic cirrhosis is caused by alcohol abuse. This problem also is the most common cause of massive upper gastrointestinal hemorrhage in the country. Laennec's (alcoholic) hepatic cirrhosis is an increasingly common cause of death particularly in middle-aged or younger males.

One of the most difficult clinical problems is the patient who has hemorrhaged secondary to esophageal or gastric varices due to portal hypertension and hepatic cirrhosis. Hemorrhage from gastroesophageal varices is usually massive. Liver function is impaired, resulting in hypoalbuminemia, which in turn affects the function of most organ systems by producing generalized tissue edema. The coagulation capabilities of such patients are disturbed with prolongation of the prothrombin time and subnormal platelet counts because of associated hypersplenism. In the presence of such major systemic problems, these patients require surgical portal decompression, a major procedure, which may be difficult and has been associated with a high mortality. Fortunately, recent improvement in the surgical management of patients who are hemorrhaging from esophageal varices and portal hypertension effectively can control bleeding with less post-operative liver failure.

RECENT DEVELOPMENTS

Recently, a number of well-controlled studies have become available regarding the long-term results of portacaval shunt for operative decompression of portal hypertension in patients who have hemorrhaged.[1,2] However, the results of portacaval shunt are already primarily of historical interest. Concurrent with the availability of final results of long-term studies, newer improved techniques for operative decompression of portal hypertension have been developed in the United States. Because of these newer procedures, developed and intensely studied within the past several years, the end-to-side portacaval shunt is considered obsolete as the operative therapy for patients who have hemorrhaged with portal hypertension.

DIAGNOSTIC PROCEDURES

Patients who have hemorrhaged and whose physical findings and history suggest portal hypertension with hepatic cirrhosis should have careful evaluation of the source of bleeding. Diagnostic techniques usually should include aspiration of the stomach by nasogastric tube and attempted demonstration of the source of hemorrhage by endoscopy. Whenever possible upper gastrointestinal roentgenographic studies should be performed. Depending on the rate of

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hemorrhage, mesenteric arterial catheterization and arteriography may be specific in demonstrating the site of hemorrhage if it is non-variceal (arterial bleeding). If the hemorrhage has stopped prior to endoscopy, the unequivocal demonstration of esophageal or gastric varices without any other associated gastric or duodenal pathology on endoscopy indicates that hypertensive submucosal gastroesophageal varices are the source of the previous hemorrhage.

NON-VARICEAL HEMORRHAGE IN THE CIRRHOTIC PATIENT

Cirrhotic patients have an increased propensity to hemorrhage from upper gastrointestinal sources other than esophagogastric varices, such as gastritis and peptic ulcer disease. Although the overall frequency of upper gastrointestinal hemorrhage from the latter sources is greater, the patient experiencing sudden, asymptomatic massive upper gastrointestinal hemorrhage in whom some stigmata of hepatic cirrhosis are present (e.g., spider angiomata, ascites, hepatomegaly, gynecomastia, or altered liver function studies) is usually hemorrhaging from gastroesophageal varices. Even before the source of the continuing rapid hemorrhage is determined, a trial of balloon tamponade should be instituted. Prompt control of bleeding usually substantiates the diagnosis of variceal hemorrhage. Balloon tamponade will not control arterial hemorrhage in the gastroesophageal region from mucosal tears, such as in the Mallory-Weiss syndrome.

RATIONALE FOR OPERATIVE PORTAL DECOMPRESSION

Patients who are known to have bled from gastroesophageal varices should undergo operative portal venous decompression to prevent further hemorrhage. The rationale for portasystemic shunt is the fact that most (65 percent) patients who have hemorrhaged from gastroesophageal varices will rehemorrhage in a period of one to two years. A high mortality is associated with the characteristically sudden and massive blood loss associated with portal hypertension. Additionally 65 to 75 percent of those patients who do rehemorrhage die from the second hemorrhage. Elective portal decompression carries only one-tenth of the mortality of emergency operation. Therefore, when a hemorrhage has been documented from gastroesophageal varices, efforts must be directed at initial control of the hemorrhage and plans for a subsequent elective portasystemic shunt.

METHODS OF TEMPORARY EMERGENCY CONTROL OF HEMORRHAGE

In patients who are hemorrhaging actively from gastroesophageal varices, temporary modalities are available to control hemorrhage initially. The most effective control measure is balloon tamponade (two pounds traction) of the gastroesophageal mucosal venous plexus. Pituitary extract (Vasopressin®) administration has been less effective but also may be a helpful adjunct. Once hemorrhage has been controlled, plans for subsequent elective portal reconstruction should be outlined specifically to the patient and his family. All concerned must understand the great potential of rehemorrhage, particularly inasmuch as the patient may be entirely asymptomatic as soon as hemorrhage is controlled. Pain is not a factor and therefore there is a natural inclination for the patient to decline future medical care.

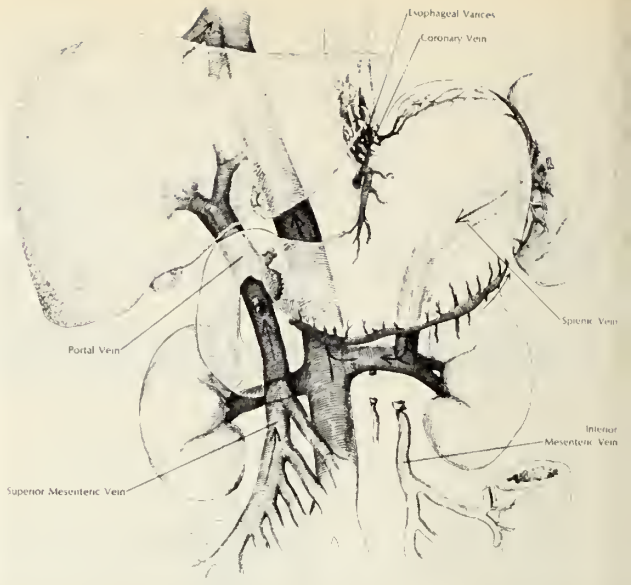


Figure 1.—*Selective Distal Splenorenal Shunt (Warren Shunt)*—In this operation, the gastroesophageal plexus is decompressed effectively via short gastric veins, through the bed of the spleen, through the splenic vein, and through the end-to-side anastomosis of the splenic vein into the renal vein. Interruption of the gastroepiploic circulation around the lesser and greater curvature of the stomach and then ligation of the coronary vein effectively segregates the decompressed segment of the portal circulation (left side) from the non-decompressed portal circulation (right side). Portal venous inflow into the liver is maintained. This may represent the basic hemodynamic reason for decreased post-shunt liver failure and post-shunt encephalopathy. (*Hospital Practice* 9:77-86, September, 1973, used with permission).

IMPROVED SURGICAL DECOMPRESSION OF PORTAL HYPERTENSION IN PATIENTS WHO HAVE HEMORRHAGED FROM GASTROESOPHAGEAL VARICES

Two improved techniques for portal venous decompression recently have become available. D. W. Warren, of Emory University developed the distal splenorenal shunt.[4] Another technique has been studied intensely and popularized by the late Theodore Drapanas of Tulane University School of Medicine.[9] This is a new type of mesocaval (H-Graft, Drapanas) shunt. Both of these new methods of operative decompression of portal hypertension now show improved long-term survival. Selective shunting is associated with a lower incidence of liver failure as compared to standard end-to-side portacaval shunts.

INDICATIONS FOR PORTASYSTEMIC SHUNTING IN PATIENTS WITH PORTAL HYPERTENSION

Portasystemic shunts may be necessary under elective or urgent conditions. An elective, *therapeutic,* portasystemic shunt is operative decompression of esophagogastric varices in patients who have hemorrhaged and who have stopped hemorrhaging. *Emergency* portasystemic shunt is performed on a patient who is bleeding actively from esophagogastric varices. *Prophylactic* portasystemic shunts are performed on patients with known hepatic cirrhosis, known portal hypertension who are not hemorrhaging actively and who never have hemorrhaged from the esophagogastric varices and portal hypertension. The current surgical management of

portal hypertension is directed at patients requiring portasystemic shunt and therapeutic portasystemic shunt. *Prophylactic* portasystemic shunts are for the most part no longer recommended.

PROGNOSIS FOR SURVIVAL AFTER PORTASYSTEMIC SHUNT

The best chance for prolonged survival lies in initial control of hemorrhage and subsequent elective portal decompression by distal splenorenal or mesocaval portal reconstruction. A number of factors enter into survival of patients with cirrhosis who require operations. Perhaps of greatest importance are the standard, time-honored classic parameters which determine cirrhotic classification (Child's).[10] These include the presence or absence of muscle wasting, encephalopathy and ascites, and the pre-operative serum bilirubin and serum albumin level. Each patient must be evaluated individually prior to operation and the vast majority of patients will enjoy prolonged survival with portasystemic decompression.

HISTORICAL INFORMATION REGARDING PORTACAVAL SHUNTS

Sophisticated clinical studies have produced general agreement regarding certain principles pertaining to portacaval shunts. Portacaval shunts effectively prevent hemorrhage from portal hypertension. Although hemorrhage is controlled, some patients develop post-shunt hepatic failure or post-shunt encephalopathy after end-to-side or side-to-side portacaval shunt. Well-designed randomized studies of portacaval shunt indicate that prolonged patient survival is achieved within some groups and subgroups of patients who have hemorrhaged and in whom hemorrhage has stopped and therapeutic end-to-side portacaval shunt is performed.[1,2] In addition there have been other well-planned studies indicating prolonged survival after portacaval shunt in patients who have hemorrhaged. For example, the controlled trial of patients, with cirrhosis and better-than-average liver functions (Child's Class A),[10] undergoing end-to-side portacaval shunt resulted in a significantly higher survival at five years after portasystemic shunt. However, all studies indicate higher incidence of encephalopathy in patients following end-to-side portacaval shunt than in non-operated cirrhotic patients who have not had major portal venous diversion from the liver.[3]

SELECTIVE DISTAL SPLENORENAL (WARREN) SHUNT[4]

Within the past decade Warren developed and studied a selective shunt to prevent recurrent hemorrhage of patients with portal hypertension and hepatic cirrhosis. Warren's shunt embodies the obvious theoretical advantage of selectively decreasing portal pressure in the gastroesophageal submucosal plexus and concomitantly maintaining portal pressure and portal flow in the portion of the portal venous tree going to the liver (Figure 1).[4] Approximately 10 years' experience has accumulated with the selective distal splenorenal shunt in patients with portal hypertension who have hemorrhaged from esophageal gastric varices. All theoretical advantages of selective distal splenorenal shunt have been realized in both hemodynamic and metabolic follow-up. Patency rate, proved by angiography, is 95 percent.[4]

Hemodynamically, selective shunting has been realized. The selective distal splenorenal shunt provides effective decompression of the gastroesophageal area by diversion of the splenic vein into the renal bed. This, in turn, decompresses the gastroesophageal mucosal plexus via short gastric veins. Concomitantly the entire mesenteric venous drainage is segregated away from the area of decompression. Therefore, the liver still enjoys perfusion of the total mesenteric venous efflux and regional portal hypertension and hepatopedal liver blood flow is maintained. The metabolic advantage of distal splenorenal shunt is believed to reside in this selective diversion of gastroesophageal area with maintenance of portal venous inflow into the liver. Thus, theoretically the liver still functions with most of the pre-operative venous blood supply, thereby maximally preserving the pre-operative hemodynamic relationships. It is, therefore, the selectivity of this shunt which provides the metabolic advantages described below.

The great advantage of selective distal splenorenal shunt in patients who have hemorrhaged from portal hypertension lies in the preservation of the liver function. Careful follow-up of increasing numbers of patients is now available. There is a definite decreased incidence of liver failure. Post-shunt encephalopathy is much less common than after standard end-to-side portacaval or side-to-side portacaval shunts.[5] This is of particular importance inasmuch as the leading

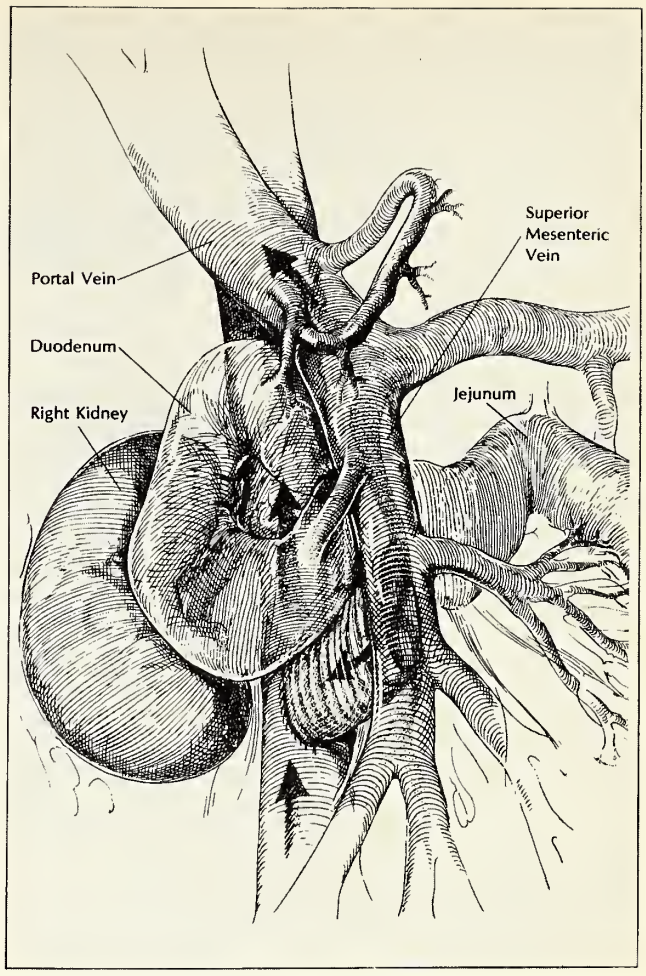


Figure 2—*Mesocaval (H-Graft, Drapanas) Shunt*—Using large bore (20-24mm) Dacron, a graft is interposed end-to-side into the superior mesenteric vein (right and above) and the inferior vena cava (left and below). Although prompt decompression of the portal venous system is obtained, the extraordinary high five-year post-shunt survival suggests that some degree of portasystemic selectivity in shunt is produced by virtue of its more distal location in decompression of the mesenteric venous system. (*Hospital Practice* 9:88-89, September, 1974, used with permission).

Table 1
Child's Classification According To Standard Clinical Laboratory Tests and Readily Observed Clinical Parameters [10]

Group Designation	"A" Minimal	"B" Moderate	"C" Advanced
Serum Bilirubin* (mg%)	Below 2.0	2.0 – 3.0	Over 3.0
Serum Albumin (gm%)	Over 3.5	3.0 – 3.5	Under 3.0
Ascites	None	Easily controlled	Poorly controlled
Neurological Disorder	None	Minimal	Advanced, "coma"
Nutrition	Excellent	Good	Poor, "wasting"

* Equivocal in biliary cirrhosis.

cause of death and morbidity following portasystemic shunt previously has been liver failure. The capability of avoiding post-shunt hepatic encephalopathy and post-shunt hepatic failure is the basis for the increased survival following distal splenorenal shunt and the decreased interest in portacaval shunts. The clinically observed lack of liver failure has been studied carefully biochemically.[5] Maximum rate of urea synthesis (capability of the liver to convert ammonia to urea) definitely is preserved in patients who have had distal splenorenal shunt as compared to surgical decompressive procedures which are less selective.[6] In our laboratory we have documented improved function of the urea cycle enzyme activities after distal splenorenal shunt as compared to portacaval shunt.[7] Our own clinical experience confirms the lower incidence of encephalopathy after distal splenorenal shunt.[8]

MESOCAVAL H-GRAFT (DRAPANAS) SHUNT

In 1975 Drapanas reported a five-year follow-up of patients with alcoholic cirrhosis who had hemorrhaged.[9] His data showed that insertion of a large-bore Dacron graft between the superior mesenteric vein and the inferior vena cava achieved effective control of bleeding. (Figure 2). Further, operative mortality is decreased dramatically. In patients with advanced cirrhosis, overall five-year survival was approximately 75 percent. Thus improved survival has been demonstrated after mesocaval shunt as well as after distal splenorenal shunt. Currently the mesocaval shunt has not been studied as carefully hemodynamically or metabolically as the distal splenorenal shunt but the definite clinical success indicates that this certainly represents an important contribution to the surgical armamentarium for use in patients who have hemorrhaged from portal hypertension. Exposure to the operative field is far superior to portacaval shunting. Blood loss is usually only a fraction of that encountered through direct portal venous reconstruction. In all likelihood the Drapanas mesocaval shunt will be the procedure of choice in those patients who require emergency portal decompression. Because this shunt decompresses the hepatic as well as the mesenteric side of the portal vein it is also effective in alleviating ascites and therefore probably will be the procedure of choice in patients who have hemorrhaged and in whom massive ascites is present.

SUMMARY

In the patient with massive gastrointestinal hemorrhage and evidence of hepatic cirrhosis, clinical appraisal, endoscopic studies, and arteriographic studies usually will document the source of hemorrhage. Hemorrhage from the esophageal varices promptly should be controlled temporarily by direct balloon tamponade. Occasionally vasopressin is given systemically, as an adjunct in obtaining temporary control of hemorrhage. Once hemorrhage is controlled, elective portal venous decompression by way of a suitable portasystemic shunt, should be planned as soon as the patient has overcome the effects of the massive hemorrhage and the frequently associated hepatic, pulmonary, and renal dysfunction associated with the sudden blood loss. If portasystemic shunt is not performed, a higher percentage of patients will rebleed and there is a high mortality associated with each hemorrhage in patients with hepatic cirrhosis. More selective surgical portal venous decompression provides effective reduction of portal pressure, effective control of hemorrhage and reduction of post-shunt hepatic failure and hepatic encephalopathy. The expected survival of patients who have hemorrhaged from alcoholic hepatic cirrhosis and portal hypertension has improved significantly.

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SPECIAL ARTICLE

Postgraduate Medical Education and Public Law 94-484

HARVEY HECHEL, M.D., East Orange*

Public Law 94-484 markedly will reduce the number of foreign medical graduates (FMGs) entering American residency programs. Seven northern industrial (snowbelt) states led by New Jersey with 68 percent FMGs in its residency programs will be affected most. In contrast, seven large sunbelt states have 9 percent FMGs in their residency programs. This manpower reduction could cripple many residency programs in high impact states.

In his book *Power Shift—The Rise of the Southern Rim*,[1] Kirkpatrick Sale clearly documents the recent, massive historical shift of economic, political, industrial, and academic power from the northern industrial states (snowbelt) to the southern and southwestern states. One aspect of this shift concerns the migration of northern physicians into postgraduate medical education in the sunbelt. During the last decade, northern residency programs, after failing to match with American graduates, filled their training programs with foreign medical graduates.

HOUSE STAFF DISTRIBUTION

Table I, adapted from data published by the AMA[2], shows the housestaff distribution as of December, 1976, for seven large northern industrial states. These states averaged almost 43 percent Foreign Medical Graduates in their residency programs. These seven states contain over 72 percent of the 15,000 foreign medical graduates presently in American residency training. This trend continued in 1977 with the snowbelt having the least success in attracting American graduates in the 1977 NIRMP match.[3]

In contrast, Table II shows the housestaff distribution in seven large sunbelt states. Foreign medical graduates average only nine percent of the housestaff in these states.

PUBLIC LAW 94-484

The Health Professions Educational Assistance Act of 1976 (Public Law 94-484) mandated that most new foreign medical graduates entering the country pass an examination equivalent to Parts I and II of the National Boards along with a rigorous English examination.[4]

During the last 10 years about 7,500 new foreign medical graduates annually filled empty residency slots. Of the 4,611 foreign medical graduates who attempted the new Visa Qualifying Examination in September, 1977, only 1,163 passed the Medical Examination. Of those 112 failed the English part.[5] Some foreign graduates who passed the VQE remain deficient in other qualifications for exchange visitor or immigrant visas and therefore were unable to come to the United States in 1978. Some of the other foreign medical graduates who passed the VQE are already in practice or residency in the United States and thus are not available to start residency programs.[6]

Last year the Secretaries of State and Health, Education, and Welfare granted a one-year blanket waiver of National Board Standards. This blanket waiver now has expired.

THE FUTURE

A reasonable estimate of annual new foreign medical

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graduates available for empty residency positions starting in July, 1979, would be about 2,500, not the previous decade's 7,500. This number will depend upon federal regulations concerning individual training programs' "substantial disruption waivers." These regulations presently are being drafted by the Bureau of Health Manpower in the Department of Health, Education, and Welfare. The final regulations will be issued by the State Department. In any event, the statute mandates that no waivers of National Board Standards can occur after 1980.

Most medical educators agree that a uniform National Board type standard for all physicians entering residency training whether American or foreign trained is a fair and justifiable goal for the medical educational system. However, as the data in Table I indicate, postgraduate residency programs in northern industrial states will be affected substantially and progessively starting in July, 1979, primarily because of Public Law 94-484.

New York, with over triple the number of foreign housestaff than any other state, particularly will be affected. Despite having 1,700 annual graduates from its 12 medical schools, New York has almost 5,000 foreign medical graduates filling about one-half of its residency training positions.

PROBLEM IN NEW JERSEY

New Jersey finds itself squeezed between the dominant cities of New York and Philadelphia in addition to having the socioeconomic difficulties of the other snowbelt industrial states. Over two-thirds of its residents (all specialties) are foreign medical graduates. New Jersey finds it difficult to attract American graduates from other states because of its direct competition with over a dozen large, well-established university centers located in New York City and Philadelphia. To many medical students, the land between the Delaware and Hudson Rivers remains *terra incognita.* This stark geographical reality is not limited to medicine. The state of New Jersey does not have a single commercial very high frequency television station; all channels are allotted to New York City and Philadelphia. Most international airlines will not land at Newark International Airport despite its prime location eight miles from Wall Street.

Most university residency programs in snowbelt states are able to attract relatively high percentages of American graduates. However, many weakly affiliated residency programs and a few university programs may be in serious difficulty within two years.

WHAT CAN BE DONE

There are two factors which may help mitigate this situation. There were 13,600 American medical school graduates in 1977. This number should increase to over 17,000 by 1982. If a residency program survives the transition period, it should have brighter prospects. American graduates of foreign medical schools who are exempted from the Visa Qualifying Examination may find themselves in strong demand. There will be a significant pool of approximately 750 to 1000 American foreign medical graduates available annually during the next five years.

There are many alternate strategies and policy options available to cope with this situation, but a discussion of these is beyond the scope of this paper. Let us hope that careful planning by medical educators in the northern industrial states will help to ease the impact of PL-94-484 on postgraduate medical education.

Table 1
Snowbelt States

State	Total Housestaff*	U.S. and Canadian Graduates	Foreign Medical Graduates	Percent of Foreign Medical Graduates
New York	9,093	4,609	4,484	49.3
Illinois	3,254	1,873	1,381	42.4
New Jersey	1,721	538	1,183	68.7
Michigan	2,755	1,725	1,030	37.4
Ohio	2,969	1,944	1,025	34.5
Pennsylvania	3,522	2,544	978	27.8
Maryland	1,830	1,151	679	37.1
Total	25,144	14,384	10,760	42.7

*Includes Interns and Residents but not Research Fellows

Table 2
Sunbelt States

State	Total Housestaff*	U.S. and Canadian Graduates	Foreign Medical Graduates	Percent of Foreign Medical Graduates
California	6,000	5,727	273	4.6
Florida	1,465	1,126	339	23.1
Georgia	930	838	92	9.9
North Carolina	1,198	1,139	59	4.9
Tennessee	988	841	147	14.9
Texas	2,735	2,432	303	11.1
Virginia	1,286	1,126	160	12.4
Total	14,602	13,229	1,373	9.4

*Includes Interns and Residents but not Research Fellows

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THERAPEUTIC DRUG INFORMATION

This information is compiled by the Schwartz Inter-National Pharmaceutic and Therapeutic Drug Information Center of the Brooklyn College of Pharmacy, Long Island University.*

1. Do you have any information concerning prazosin in the treatment of congestive heart failure?

Vasodilator therapy is emerging as an important adjunct in the management of congestive heart failure. Prazosin (Minipress®)was introduced recently as an antihypertensive and is currently under investigation for the treatment of heart failure. The drug dilates both the arterial and venous beds and has no significant effect on cardiac output or reduction of renal blood flow.[1]

Awan *et al.*[2] studied the effects of oral prazosin on the peripheral circulation in ten subjects, including seven patients with chronic congestive heart failure due to coronary disease. Prazosin was demonstrated to induce systemic venodilation and peripheral arteriolodilation. These pharmacological effects indicated that the drug had potential benefit for relief of pulmonary congestion and elevation of cardiac output in patients with chronic congestive heart failure.

Another study[3] evaluated the long-term efficacy of prazosin in nine patients with refractory heart failure due to chronic coronary disease. Symptomatic evaluation was carried out for two to four months at weekly intervals. One hour following two to seven mg of prazosin, left ventricular filling pressure was reduced, and this effect remained for a six-hour period. Patients were asymptomatic throughout the duration of follow-up. The authors concluded that prazosin possessed sustained nitroprusside (Nipride®)-like actions on the systemic arterial and venous systems, and is effective in the management of ambulatory patients with chronic severe heart failure.

A comparison of cardiocirculatory actions of nitroprusside to prazosin was conducted in 11 chronic coronary patients with refractory congestive heart failure.[4] Both drugs produced an equal but mild systemic lowering of arterial pressure without alteration of heart rate. Both drugs also equally enhanced efficiency of cardiac output and myocardial oxygen consumption index. Total systemic vascular resistance declined equally with both drugs. It was concluded that since prazosin produced six hours of symptomatic improvement, it is the best vasodilator for long-term use, and extends in-hospital nitroprusside-like action to ambulatory heart failure therapy.

Miller *et al.*[5] studied the effects of prazosin in ten patients with ischemic cardiomyopathy and severe ventricular dysfunction and reported that prazosin was beneficial in the treatment of severe congestive failure.

In conclusion, oral prazosin appears to be an effective adjunct in the management of chronic congestive heart failure, but further studies are needed to define its ultimate role.

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2. Please provide me with information concerning ticrynafen (Selacryn®) in the treatment of hypertension.

One of the disadvantages of thiazide and other potent diuretics is the possibility of hyperuricemia, which may precipitate gout. Ticrynafen, a newly synthesized diuretic, currently under investigation by Smith, Kline, and French Laboratories in the United States, appears to combine the diuretic and antihypertensive properties of thiazides with potent uricosuric effects.[1]

Nemati *et al.*[1] conducted a double-blind randomized study of ticrynafen in 20 hypertensive patients. The patients were assigned to a six-week regimen of either ticrynafen (one tablet daily, 250 mg) or hydrochlorothiazide (one tablet

*The Center serves as a source of intelligence on therapeutic and pharmaceutic information not readily available to physicians, at no charge to them, and provides this information with minimal time involvement. It is staffed by trained pharmacists; Jack M. Rosenberg, Pharm.D., Associate Professor and Chairman, Division of Clinical Pharmacy, Brooklyn College of Pharmacy, is Director and Walter Modell, M.D., Emeritus Professor of Pharmacology at Cornell University Medical College, is pharmacologist consultant. The service is available Monday through Friday from 9 a.m. to 4:30 p.m.—telephone (212) 622-8989 or 303-2735. Responses to these questions were prepared by J. M. Rosenberg, M.S.; Pharm.D.; D. Sherman, M.S., P. Sangkachand, M.S., R.Ph.; Doris Lau, B.S.

daily, 50 mg). Blood pressure was significantly reduced with both medications. Whereas the serum uric acid level rose moderately in the hydrochlorothiazide-treated patients, it fell strikingly in patients treated with ticrynafen. The authors concluded that ticrynafen appears to be a useful new antihypertensive agent because of its unique combination of diuretic, antihypertensive, and hypouricemic effects.

Carvalho *et al.*[2] conducted a crossover clinical study comparing ticrynafen, 250 mg twice daily, with hydrochlorothiazide (HydroDiuril®, Oretic®, Esidrix®, and others), 50 mg twice daily, in 12 hypertensive patients. Both agents significantly decreased mean arterial pressure, but ticrynafen was associated with reduction of serum uric acid.

Stote *et al.*[3] conducted a crossover design study to compare the effects of ticrynafen with several other diuretics including chlorthalidone (Hygroton®), ethacrynic acid (Edecrin®), furosemide (Lasix®), and hydrochlorothiazide. The authors found that over an eight-hour period, a 250 mg dose of ticrynafen exerted natriuretic activity comparable to that of 25 mg of chlorthalidone and 50 mg of ethacrynic acid; a 500 mg dose of ticrynafen was comparable to 50 mg of chlorthalidone and 50 mg of hydrochlorothiazide. Ticrynafen combined with furosemide had an additive diuretic effect, whereas in combination with hydrochlorothiazide it was not additive. Ticrynafen exhibited uricosuric activity and this effect was maintained when given with furosemide and hydrochlorothiazide, the latter two individually were slightly antiuricosuric.

In conclusion, ticrynafen may be useful in patients requiring a diuretic in whom it is desirable to avoid hyperuricemia.

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3. Is there an interaction between phenylbutazone and oral contraceptives?

Phenylbutazone (Butazolidin®) is among the drugs which have been proved in animals to accelerate, via induction of hepatic microsomal enzymes, the biotransformation of steroids, including those which are constituents of oral contraceptives.[1] As a result of this accelerated biotransformation, the effects of an oral contraceptive may be reduced.

That phenylbutazone possibly may stimulate metabolism of oral contraceptives in humans, in addition to animals, is suggested in many drug interaction reference sources.[2-7] The consequences of this interaction that have been proposed are spotting, breakthrough bleeding, or in the extreme case, lack of effective contraceptive activity.

According to Hempel and Klinger,[1] steroid-inducing effects of phenylbutazone have not been proved in humans, but nevertheless, they recommend that if bleeding disturbances should occur (for example, spotting or breakthrough bleeding), this method of contraception should not be relied upon.

Our search of the literature did not reveal any reports in humans of spotting, breakthrough bleeding, or pregnancy resulting from an interaction between phenylbutazone and oral contraceptives.

In conclusion, although phenylbutazone has been shown to interact with oral contraceptives in animal experiments, we could not find any documented reports of this interaction occurring in humans.

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PEDIATRIC BRIEFS

Selected Abstracts with Comments*

Limitation of Sore-Throat History as an Indication for Tonsillectomy. Paradise JL, *et al: N Eng J Med* 298:409, 1978.

As part of an ongoing study of the indications for T and As, 65 children with "impressive" histories of recurrent throat infections ("impressive" defined as at least seven episodes in one year, five in each of two consecutive years, or three episodes in each of three consecutive years) were followed with a detailed protocol of frequent interviews and examinations for an additional two years. The original histories were not well documented. Only 11 children (17 percent) repeated their own historical patterns while under observation; thus 83 percent were not considerad candidates for T and As. No statistically significant characteristics (such as age, sex, socioeconomic status, or "reliability") identified the repeating 11 percent.

Comment: This impressive study can be responded to in one or both of two way:(1) it provides valuable hard data with which to support anti-T and A "arguments" with surgeons, hospital administrators, *and* parents; (2) it strongly suggests that watchful waiting is the preferred course for any child with a "bad" year, or two, of sore throats, especially if these are undocumented. (N. Sissman, M.D.)

Nonoperative Management of Traumatized Spleen in Children: How and Why. Ein SH, *et al: J Ped Surg* 13:117-119, 1978.

The authors state that people without spleens have a 50 times greater incidence of overwhelming sepsis than normal, that such sepsis has a 50 percent mortality rate and that susceptibility remains throughout life. Nonoperative management for traumatized spleens has been emphasized at the Toronto Hospital for Sick Children for 35 years. Their regime is: place patient in ICU, insert nasogastric tube and large bore IV. Splenic scan usually shows filling defect in damaged spleen. If in "mild" shock with Hgb more than 10 gms percent, watch and give fluids; if Hgb less than 10, transfuse. If BP and Hgb remain "unstable," or if there are signs of increasing intraabdominal bleeding (mainly abdominal distention and increasing abdominal tenderness) operation is imperative. Between 1972 and 1977, 56 children with proved damaged spleens were treated. 21 were operated upon; 35 were not—all without mortality. In 1976-77, only one child was operated upon. Successful non-operatively treated patients usually began to improve within 24 to 48 hours. They remained hospitalized for 2 to 3 weeks; restriction of activity for up to two months and repeated scans to document healing are important management components.

Comment: These results are to be emulated. Success clearly requires very close observation and around-the-clock surgical and blood bank back-up. Another article in the same issue of that journal describes techniques of laceration repair, partial splenectomy and even re-implantation of pieces of spleen to avoid the consequences of total splenectomy.

(N. Sissman, M.D.)

Comment: The series of studies now reported are all coming to the same conclusion. Splenectomy needs to be done in the *minority* of patients with splenic rupture. The vast *majority* either can be repaired or treated supportively.

(R. H. Rapkin, M.D.)

Care of Infants with Near-Miss "Sudden Infant Death Syndrome." Kelly DH, et al: *Pediatr* 61:511-514, 1978.

"Near-misses" are babies who are resuscitated successfully after an apneic episode. Since there is a 20 to 100 percent chance of mortality from crib death following such an episode, these babies require aggressive management.

Sixty infants (average gestational age 39 weeks, average birth weight 3.1 kg) resuscitated after severe apnea associated with cyanosis were placed on apnea monitors at home. Twenty-six of these monitored infants had apneic episodes of sufficient severity to demand bag and mask resuscitation which was successful for every initial episode. However, four infants died during subsequent apneic attacks mainly because the parents did not hear the alarm. This represents a 6.6 percent mortality rate as compared to the probable 43 percent mortality rate (26/60) if monitoring and resuscitation had not been provided.

Comment: The authors conclude that most deaths in these babies can be prevented by a program of home use of apnea monitors coupled with appropriate intervention by parents trained in resuscitation. (R. Meny, M.D.)

Hand-to-Hand Transmission of Rhinovirus Colds. Gwaltney JM, et al: *Ann Intern Med* 88:463, 1978.

Volunteers were infected experimentally with Rhinovirus (RV). Sixty-five percent of infected patients had virus on

*Abstracted from "Pediatric Department Newsletter," RMS, Vol. 2, No. 9 (May 1978). Selections are made and original comments prepared by Richard H. Rapkin, M.D., Professor of Pediatrics, RMS, CMDNJ, and his associates.

their hands. Forty-five percent of hand contact recipients had virus on their hands. Viral recovery from coughs and sneezes was low. Hand contact-self inoculation may be the most important route of contagion of common cold.

Comment: Although not the only route of transmission, hand contact may be a major, if not the major route. If that is so, efforts to alter our habits may reduce transmission. We all have hand-to-face unconscious mannerisms. We usually fail to wash our hands after contact with our faces or others' hands. We shake hands too much. (R. H. Rapkin, M.D.)

Relation of Breast Versus Bottle Feeding to Hospitalization for Gastroenteritis in a Middle-Class U.S. Population. Larsen SJ et al: *J Pediatr* 92:417, 1978.

In a closed-group practice 34 of 35 admissions to the hospital for gastroenteritis were in bottle-fed infants. Twenty-eight percent of infants in the practice were breast-fed.

Comment: This little study supports much other data. In addition, it demonstrates that very valuable research can be done in office practice. Anyone interested?

(R. H. Rapkin, M.D.)

Clinical Diagnosis of Inborn Errors of Metabolism in the Neonatal Period. Nadler HL, et al: *Pediatr* 61:398, 1978

Neonates with metabolic errors may present with unexplained vomiting, diarrhea, jaundice, seizures, hepatomegaly, or with hypoglycemia or metabolic acidosis. Simple diagnostic tests are easily done. Confirmatory tests can be done later.

Comment: This is an important article which should be available in the nursery for ready reference. Although individually rare, these disorders are collectively not uncommon. Many are treatable. All have major genetic counseling implications. (R. H. Rapkin, M.D.)

Social and Medical Prognosis of Children with Acute Purulent Meningitis. Sillanpaa M, et al: *Acta Ped Scand* Suppl. 265, 1977.

Two hundred forty-four patients with bacterial (nontuberculous) meningitis from 1948-1972 were studied. Twenty-three percent died of the disease, 76 percent were completely normal, 17 percent had mild or moderate disability, 7 percent have severe disability. The prognosis was related to age; 80 percent of infants, 85 percent of children, but only 72 percent of neonates were normal on follow-up. The organism also "counted": 80 percent of hemophilus, 93 percent of meningococcus, and 80 percent of pneumococcus were normal on follow-up compared to 65 percent of E. Coli.

Comment: This comprehensive study provides most definitive answers to the prognosis questions. Of patients who survive meningitis 76 percent will recover completely. The majority of the remainder will be able to function independently. Although no cause for celebration, this could provide a baseline for our efforts. Obviously the real need is prevention and the development of vaccines which will be immunogenic in infancy. (R. H. Rapkin, M.D.)

The Extent and Character of Drug Consumption during Pregnancy. Doering PL, et al: *JAMA* 239:843, 1978.

All prenatal patients studied received at least two drug products during pregnancy (iron, analgesics, vitamins, topicals, antacids, cough remedies, laxatives, antibiotics, antiemetics, and others) with the average being eleven!! Many of the products contain multiple ingredients. Most of these drugs have not had adequate testing to assure safety in pregnancy.

Comment: Evidence again that we are somehow secure in our role as healer despite the fact that history shows most healers of the past have done more harm than good.

(R. H. Rapkin, M.D.)

High-Fiber Diets in the Treatment of Diabetes Mellitus. Miranda PM, et al: *Ann Int Med* 88:482, 1978.

Dietary fiber reduced mean plasma glucose from 170 to 120. It reduced insulin need and occasionally (if insulin was not reduced) caused hypoglycemia. Variation in dietary fiber by the diabetic may influence control and insulin need.

Comment: Dietary fiber may help to control diabetic glucose fluctuations and may account for problems of understanding spontaneous fluctuation in control.

(R. H. Rapkin, M.D.)

Hepatitis A Virus Infection. Dienstag JL, et al: *J Infect Dis* 137:328, 1978.

The prevalence of HAV increases with age, decreases with class, is enhanced by poor personal hygiene. There is no increased incidence in homosexuals, among hemodialysis patients, or multiply transfused individuals. Viremic or intestinal *carriers* occur rarely if at all. HAV rarely if ever is spread parenterally. It plays no role in chronic liver disease and rarely causes fulminant hepatitis.

Comment: HAV infection is now diagnosable serologically. With these powerful tools the above information was generated. Although Hepatitis B is a problem both parenterally and enterally, HAV appears to be transmitted only during the acute illness (including incubation period) and only enterally. However HAV accounts for a sizeable proportion of adult hepatis (20-40 percent).

(R. H. Rapkin, M.D.)

Housing Application

213th Annual Meeting

The Medical Society of New Jersey

May 12-15, 1979

	DAILY RATES		
	Single	Twin	Suites
Holiday Inn	$42	$46	Rates quoted upon
(MSNJ Headquarters)	50	54	request
	58	62	
Howard Johnson's	$42	$46	Rates quoted upon
(Auxiliary Headquarters)	50	54	request
	58	62	

If room is not available at rate requested, next available rate will be assigned

Mail this application directly to the Atlantic City Convention Bureau
16 Central Pier
Atlantic City, N.J. 08401

Please list 1st and 2nd choice; confirmation will come directly from hotel.

1st Choice.....2nd Choice.....

Accommodations desired: ☐ Single ☐ Twin ☐ Suite Parlor & 1 Bedroom
☐ Suite Parlor & 2 Bedrooms

Name.....

Address.....

City..... State..... Zip.....

Phone.....

Will arrive..... Time..... Will depart..... Time.....
DATE DATE

☐ Check if Official Delegate..... County

Trustees' Minutes November 19, 1978

A regular meeting of the Board of Trustees was held on Sunday, November 19, 1978, at the Executive Offices in Trenton. Detailed minutes are on file with the secretary of your county medical society. A summary of significant actions follows:

Conference with HEW Secretary Califano . . . Received as informative a written report from President Krueger on the press conference with HEW Secretary Califano relative to special federal funding to combat cancer in New Jersey. Mr. Califano has announced that the four-point program:

1. Will create the first statewide anti-smoking program aimed at getting adults to quit and teenagers never to start; $100,000 of federal money will be forthcoming for this.
2. Will give $360,000 over a two-year period to fund a portion of the New Jersey Subcommittee for the Control of Cancer.
3. Will give $930,458 over a three-year period to the New Jersey State Department of Health to establish a statewide cancer of the cervix screening program.
4. Will give over $1,000,000 over a four-year period to document the case histories of 4,000 patients who have cancer. Occupational history will be closely reviewed, along with possible environmental factors. Patients studied will be primarily from Cumberland, Gloucester, Salem, Hunterdon, and Warren Counties because of the high reported incidence of cancer in these counties.

Antitrust Suit against the AMA and others by the Pennsylvania Chiropractic Society . . . Agreed to support the current position of the AMA regarding settlement of an antitrust suit brought by the Pennsylvania Chiropractic Society.

. . . Agreed to the position of the AMA and the Pennsylvania Medical Society's Board of Trustees of "accepting the fact that chiropractors are licensed, limited practitioners in Pennsylvania and that physicians may or may not choose to accept patients sent to them by chiropractors, or anybody else."

. . . Noted that the Pennsylvania Medical Society's House of Delegates, meeting October 30 to November 1 stated: "If approved in the manner in which it was presented to the courts, the Pennsylvania Medical Society would not be admitting guilt to any action and it would still maintain the right to question the unscientific benefits of chiropractic." It was anticipated that settlement of this suit in Pennsylvania will have an effect on similar lawsuits in New Jersey and Chicago. The legal issue as well as the principles of medical ethics surrounding this settlement will be discussed at the forthcoming Interim Meeting of the AMA (December). MSNJ's Board of Trustees favored continued study by the AMA Ad Hoc Committee on the Principles of Medical Ethics.

Headquarters Mortgage Financing . . . Approved the concept of the mortgage financing proposal presented by Mr. Maressa, Executive Director, and authorized the executive staff to submit a definitive report of the recommended financial arrangements for Board sanction.

MSNJ Student Association . . . Noted that membership in MSNJ's Student Association is currently 165, the majority of whom are freshmen. In accordance with the action of the 1978 MSNJ House of Delegates, the membership fee for students is $5 a year, which includes subscription to *The Journal.*

New Jersey State Medical Underwriters, Inc.:

1. Claims Committee and Peer Review Mechanism . . . Noted that 479 cases were reported, of which 48 percent were indefensible and 52 percent defensible; 94 cases were closed, 45 of which were peer-reviewed; 385 cases are outstanding, 111 of which have been peer reviewed.

. . . Approved a recommendation that the peer review program as established by the Underwriter be continued indefinitely with annual reports (and additional reports as may be requested) to be made to the Committee on Medical Defense and Insurance.

2. Professional Liability Coverage . . . Noted that the New Jersey Hospital Association-sponsored Health Care Insurance Exchange has agreed to provide primary liability coverage for physicians engaged in hospital committee and related staff work. MIIE's policy will provide standard professional liability insurance for similar activity outside the hospital. Presently Medical Underwriters must secure this type of coverage from other major insurers of hospitals in the state and the individual hospitals who are insured by other carriers.

3. Umbrella Coverage . . . Received the following communication from the president of New Jersey State Medical Underwriters concerning umbrella coverage for liability not presently insured:

"The Exchange has examined this issue thoroughly and concludes that such coverage is not feasible at this time. The problem centers on the issue of whether or not it is legal or proper to relieve an insured doctor of responsibility for willful acts which are in violation of the statutes. The unfortunate difficulty for a physician named in a complaint, dealing with his committee or staff involvement, is the fact that such a complaint often contains allegations of conspiracy (or some other malicious intentional act) and demands punitive damages. It is against public policy in New Jersey for an insurer to relieve any malfeasor of the financial impact of an adverse finding calling for the payment of punitive damages.

"The Exchange assures its policyholders that should demands for punitive damages be included with any charge of liability—stemming from the insured's professional activities covered under the Exchange's policies—defense on both issues will be provided. The company is only permitted, however, to pay compensatory damages arising from the case.

"This may sound like an all-too-technical, negative response to a straight-forward question. Our problem is that, unless regulatory conditions change, your company will be unable to fill what you consider a basic insur-

ance need. The Underwriter staff will continue to watch the situation and review the issue once conditions change."

4. Department of Liability Control . . . Noted that the Professional Liability Fund shows a balance of $1,038,766.55 as of September 30, 1978, which will be used to educate physicians in New Jersey in how to avoid the type of problems which the Medical Inter-Insurance Exchange of New Jersey now is handling.

. . . Meetings are planned with specialty societies to present more information concerning professional liability as it relates to that particular specialty, and it is anticipated that legal bulletins will be published for these societies.

5. Donald F. Smith and Associates . . . Decided to postpone further discussion, until the anniversary date of the program (June 1979), of alternate rate schedules for the Major Medical Program which Blue Cross/Blue Shield would be willing to offer with substantially lower rates but drastic reductions in benefits.

Medical Inter-Insurance Exchange Rate Revision . . . Noted that MIIE had filed a rate revision that: (1) will not increase premiums for anyone; (2) will reduce premiums for those classes whose experience is most favorable; (3) accurately will reflect experience by realigning classifications; and (4) will use what is left of the overall premium reduction to diminish the degree of subsidization in the program.

Impaired Physicians . . . Received a report from the Committee on Impaired Physicians which noted that all is in readiness for implementation of the program contingent upon completion of an informational pamphlet, which has been discussed in its first draft, and participation in a discussion relative to the legal implications of the Committee's activities.

Emergency Medical Care . . . Received as informative a report from the Special Committee on Emergency Medical Care which noted that the American Medical Association/American Hospital Association Invitational Conference on the Categorization of Hospital Emergency Capabilities had set up twelve task forces to discuss and exchange ideas on the categorization of hospitals. Guidelines presently oriented toward trauma will be directed in the future to all emergencies.

Medicaid Committee Meeting with Representatives of the Division of Medical Assistance and Health Services . . . Received as informative a report of the joint meeting of the Committee on Medicaid with representatives of the Division of Medical Assistance and Health Services which considered among other things:

1. The problem encountered with pharmacists who will fill a prescription under the Pharmaceutical Assistance to the Aged Program for only a month's supply, and, if necessary to increase dosage which would exhaust the patient's medication before the end of the month, the patient is billed for an additional prescription. The representative of the New Jersey Pharmaceutical Association indicated that federal and state law permits a 60-day supply for dispensing drugs, and that the pharmacist cited is being overly conservative.

2. That an ad hoc committee of MSNJ and Medicaid concerned with revision of regulations will explore the mechanism for a dialogue between the profession and Medicaid on updating the regulations, specialty by specialty.

Medicaid Reimbursement . . . Received as informative a report from the Executive Director on a meeting of the Executive Committee with members of the Governor's staff concerning provider reimbursement for Medicaid patients, which noted that the Governor's Counsel, Mr. Pollock, would like to put an end to the hostility and proceed to resolve the issues; that the Governor had notified his cabinet that he wanted the budget cut and not expanded; that if the Governor were in a position to expand the budget, he would appropriate funds in the health care provider area; that if it can be demonstrated that a private physician's office is more efficient and less expensive than a hospital outpatient clinic, steps will be taken to encourage patients to use the physician's office. The Executive Committee had granted Mr. Pollock and his staff thirty days to review what can be accomplished with this approach.

Note: It has been rumored that physicians may elect not to participate in Medicaid meetings because of alleged harassment by Medicaid surveillance. It was suggested that such rumors quickly be dispelled or verified as factual, and further suggested that a poll be taken of all those who have attended the committee meetings to determine their rate of audit as compared to that of the general population of physicians.

Board of Medical Examiners . . . Received as informative reports from Arthur Bernstein, M.D. (October 18th meeting) and from Frank Campo, M.D. (November 19th meeting) on the State Board of Medical Examiners' meetings, and noted that the Board had been requested to provide (for the MSNJ representatives) an agenda and materials in advance of the meetings, if available.

At the October 18th meeting, the President of the State Board of Medical Examiners made the statement that professional fees are much too high, and appointed a committee to meet with various societies so that a rule can be promulgated to control fees in New Jersey. When pressed for specifics, he refused on the grounds that it was privileged information. When challenged on the fact that there are judicial committees whose duties include looking into such matters, the President of the State Board of Medical Examiners observed, as his opinion, that the judicial committees were not effective and were not doing a good job to bring fees under control.

. . . Directed that the State Board of Medical Examiners be advised that MSNJ is willing to cooperate and desires to meet with the subcommittee as an approach to reaching a solution to the alleged problem of excessive fees. It is to be pointed out to the State Board of Medical Examiners that the MSNJ mechanism, working within the confines of the law, has been quite successful when properly utilized by patients, insurance companies, and State agencies.

. . . Directed that the president of the Union County Medical Society, who expressed concern over the State Board of Medical Examiners' airing fee disputes relative to medical practice and castigating the entire medical profession, and criticizing the judicial mechanism, especially in view of the fact that the powers of the judicial committee have been limited by the State Board of Medical Examiners and other agencies, be advised that the Board of Trustees has offered to cooperate with the Board of Medical Examiners on the above-named committee.

Health Care Administration Board . . . Received a written report from MSNJ's representative to the Health Care Administration Board (Daniel J. O'Regan, M.D.) and a draft of the Proposed Emergency Medical Services' Regionalization Standards, and directed

that the proposed standards and comments from Rudolf E. Schwaeble, M.D., president of the New Jersey Chapter, American College of Emergency Physicians, be referred to MSNJ's Committee on Emergency Medical Care and the Committee on Trauma of the New Jersey Chapter, ACS, for review and comment. Dr. Schwaeble called attention to the fact that the New Jersey Emergency Medical Services Council was not consulted in drafting such standards, and that the Emergency Medical Services Systems Act of 1973 (P.L. 93-154) provides that the Council's participation is a condition specified by HEW for the Department of Health to receive grants for emergency medical services' regionalization projects within the State.

Model Health Care System . . . Received as informative a report from the Task Force to Review the New Jersey Department of Health Model Health Care System, which elicited the following: There are problems in the health care system. There is needless waste, over-utilization, under-utilization, and misutilization. Quality is divergent, rarely uniform, and difficult to measure. In spite of success, there is room for improvement. In spite of failures, it is the best system available. Though the altruistic motivation of those rendering the services may be challenged, we indeed have the most cost-efficient system in the world.

The Model Health Care System—which is utopian in concept and not grounded in reality, and for which no strategy is outlined for implementation—deals with three concepts:

1. Resources and services—which provide cradle to grave universal coverage for all persons. It would relocate physicians and patients alike and evolves into a highly centralized, socialized welfare state system that is not concerned with quality. Students selected to enter medical training will be chosen not on the basis of academic achievements, but according to "other personal qualities."

2. Administration and Regulation—there will be total central administration to determine how services are to be provided. There will be no consideration for experience, motivation, or initiative.

3. Financing: There is envisioned a mandatory universal health insurance plan using a combination of general taxes and employer/employee payroll taxes. Supplemental benefits could be purchased from private insurers. The disburser would be either a public authority or fiscal intermediaries. Emphasis would be on primary care, early treatment, and outpatient services. Inpatient services would be discouraged.

Voluntary Cost Containment . . . Voted to support the following eleven recommendations as contained in the report of the Voluntary Cost Containment Committee of New Jersey:

1. Support the efforts of the New Jersey Department of Human Services to obtain a waiver from the Department of Health, Education, and Welfare to allow hospitals to be paid by the Medicaid Program appropriate long-term care rates for those patients who no longer require acute care services but for whom no long-term care beds are available. In addition, there should be a waiver mechanism established by the New Jersey Department of Health permitting hospitals to convert voluntarily unneeded acute care beds to long-term care beds.

2. The Department of Health, Education, and Welfare (HEW) and the Office of Management and Budget should examine the issues relative to the End-Stage Renal Disease Program and remove from the program all duplicative and counterproductive regulations and reporting requirements. If necessary, they should recommend legislative modifications to achieve the least costly and most efficient data-collection reporting and processing system.

3. HEW should suport the gradual expansion of the mandate of Professional Standards Review Organizations initially to include monitoring the care received by *all* hospital patients, and later, the care rendered in all sites for health care delivery. New patient care review activities should be phased in only after their effectiveness has been evaluated and demonstrated, and any new activities should be funded adequately.

4. HEW should require Professional Standards Review Organizations to report annually and make public their status relative to relationships with physicians and institutions and their status and coordination with other regulatory bodies. These reports should preserve the confidentiality of identifiable personal health-related information in the absence of an authorized release of such information by each affected patient, practitioner, and institution.

5. As a primary cost-containment effort, the New Jersey Hospital Association, as a top priority, should have a strategy attacking statutes and regulations which interfere with the governance structure and day-to-day management of hospitals. These laws are generally costly, unnecessary, and have little or no effect in enhancing quality of care.

Such a strategy should include the following:

a. Arrange for discussions between interested parties and agency staff during regulation development.

b. Provide staff regulations and policies to interested parties for review prior to publication.

c. Review regulations after they are implemented to check on their effectiveness.

6. As a major cost-containing effort, support the American Hospital Association's position that it should continue to maintain and strengthen its capability to monitor the development of legislation and regulations, develop legislative proposals, monitor and analyze the matters involved, inform and, when appropriate, counsel allied associations and membership, and develop and present appropriate comments and recommendations to Congress and regulatory agencies.

7. Support the American Hospital Association's Board of Trustees' recommendation that all hospitals and allied hospital associations should participate actively in the American Hospital Association's Partnership for Action Program in order to implement fully and improve hospital participation in the legislative and regulatory process.

8. Support the American Hospital Association's Board of Trustees' recommendation that all hospitals and allied associations should work actively with the American Hospital Association to analyze the impact of regulations and bring to the attention of appropriate agencies instances where the regulations have had adverse effects and propose recommended solutions.

9. The Commissioner of Insurance should provide that insurance companies use the Building Officials and Code Administrators Basic Building Code (BOCA) rather than the National Building Code. BOCA Basic Building Code is more consistent with hospital Life Safety Code standards.

10. All hospital licensure standards for the Department of Health should be revised to conform with state and federal laws and regulations. This action would eliminate some critical conflicting standards that are currently applied to hospitals.

11. The Medical Society of New Jersey and NJHA should explore the cost-restraining potentials of the "physician extender" concept.

Physicians' Assistants . . . Defeated a motion to rescind the action of the Board of Trustees taken on October 15 in giving "Conditional Approval" to bills S-1192 and A-1387 which provide for certification of physicians' assistants. (MSNJ continues to oppose A-1314)

. . . Directed, in view of petitions received from seven county medical societies, that a special session of the House of Delegates be convened on December 10 at Rutgers Medical School.

. . . Defeated a motion for another poll of the membership on the issue of physicians' assistants.

Dean of Rutgers Medical School . . . Voted unanimously to submit the name of Palma E. Formica, M.D., as the Society's recommendation for appointment to the search committee to seek qualified candidates for the position of Dean of Rutgers Medical School, CMDNJ.

House of Delegates Special Session December 10, 1978

Two hundred and ninety-two delegates from twenty-one county societies and seventeen fellows and officers of the Medical Society of New Jersey participated in the Special Session of the House of Delegates, held on Sunday, December 10, 1978, in Piscataway, to consider the action of the Board of Trustees on October 15, 1978 in relation to physicians' assistant legislation (A-1387 and S-1192) and matters referring thereto. The delegates were supplied with relative materials in advance of the meeting.

The session was called to order at 10:10 a.m. by Charles S. Krueger, M.D., President. The invocation was delivered by Watson E. Neiman, M.D., Delegate from Burlington County. The Chairman of the Committee on Credentials, Charles I. Nadel, M.D., reported that a quorum of the delegation was present. The meeting then was turned over to the Speaker, Henry J. Mineur, M.D.

By a voice vote, the House of Delegates agreed to admit members of the press to the session.

The House, also by voice vote, agreed that no one be permitted to speak more than once (except in rebuttal) on any given subject, except by permission of the House, and that a speaker's time be limited to four minutes.

James S. Todd, M.D., Chairman of the Board of Trustees, was given the floor, to make a presentation summarizing the purpose of the special session and supporting the legality of the Board's action pertaining to the physicians' assistant legislation.

Doctor Todd noted that evidence presented to the Board of Trustees by outside consultants and staff included:

(1) Overwhelming media and press support for the passage of the physicians' assistant legislation.

(2) All major agencies of the State Government, including the State Board of Medical Examiners, have voted to support the bills and are aggressively seeking passage.

(3) No visible legislative support to sustain MSNJ's continued opposition.

(4) Clear indications that the majority of practicing physicians favor being granted the legal right to decide for themselves whether to employ such personnel.

Confronted with the foregoing, the Board voted to **conditionally approve** A-1387 and S-1192 with suggested amendments offered to assure quality of care and cost effectiveness.

The Speaker opened the floor for discussion at 10:45 a.m. Discussion terminated at 12:10 p.m.

By a show of hands' vote, the action of the Board of Trustees in relation to the legislation (A-1387 and S-1192) was not supported by the Delegates.

The House voted to oppose A-1387 and S-1192 actively, and to inform the legislature that licensure and/or registration of physicians' assistants will neither contribute to the quality nor decrease the cost of medical care.

Frank Y. Watson, M.D., Delegate from Essex County, and Chairman of JEMPAC, presented the following Plan for **Active Opposition** to Physicians' Assistant Legislation (A-1387 and S-1192):

ROLE OF THE MEDICAL SOCIETY OF NEW JERSEY

1. Send letters to all legislators indicating active opposition and reasons for it.
2. Prepare a news release to all media indicating active opposition and reasons for it.
3. MSNJ Trustee Delegation and MSNJ Lobbyists visit key legislators and the Governor indicating active opposition and reasons for it.
4. Coordinate lobbying effort with other organizations including the New Jersey Academy of Family Physicians and the New Jersey State Nurses' Association.
5. Activate the Keyman Program.
6. Members of the House of Delegates in each Legislative District join keymen as a delegation and visit the legislators in their district indicating active opposition and the reasons for it and ask the legislators for their support.

ROLE OF COUNTY MEDICAL SOCIETIES

1. Coordinate lobbying of keymen and members of House of Delegates in their county.
2. Ask members to send letters to their legislators and the Governor indicating active opposition and reasons for it.
3. Establish a Trenton Political Task Force of physicians and auxillians who, on short notice, will lobby their respective legislators in their home district and in Trenton when the physicians' assistant legislation is being considered.
4. Send a delegation to the State Board of Medical Examiners indicating active opposition and the reasons for it.

The House of Delegates adopted Doctor Watson's plan.

A resolution pertaining to the Medicaid program was introduced by Harvey J. Shwed, M.D., Delegate from Essex County, and Seymour Charles, M.D., Alternate Delegate from Essex County. However, the resolution failed to receive the consent of a majority of the delegates for discussion.

The meeting of the Special Session was adjourned by Doctor Krueger, at 12:35 p.m.

Supreme Court Rule

Rule 4:21. Professional Liability Claims Against Members of Medical Profession: Procedure

4:21-1. Purpose of Rule

The procedure set forth in R. 4:21 is for the common interest of the public, the medical and legal professions in the processing of medical malpractice actions with the view toward discouraging baseless actions and encouraging settlement of those actions based on reasonable medical probability; to monitor efficiently these cases through the court; and, to assist in the early disposition of medical malpractice actions.

Note: Adopted July 24, 1978 to be effective September 11, 1978.

4:21-2. Processing of Medical Malpractice Cases

(a) **Caption of Action.** A medical malpractice action shall be so designated in the caption and be given a special identifying letter by the clerk.

(b) **Judicial Assignment.** The Assignment Judge shall, whenever feasible, designate one judge before whom all medical malpractice cases in his vicinage shall be pretried. The judge so designated shall preside at all pretrial discovery proceedings, pretrial conference, and the hearing set forth herein but shall not preside at the trial of the action.

(c) **Pretrial Conference.** The court shall conduct a pretrial conference pursuant to R. 4:25-1 not later than twelve months following service of process upon the defendants. The pretrial order shall provide for the submission of the claim to a panel as hereinafter provided and shall prescribe a schedule for further processing of the panel hearing.

Note: Adopted July 24, 1978 to be effective September 11, 1978.

4:21-3. Panel Members: Doctors and Lawyers

For purposes of Rule 4:21, the Administrative Director of the Courts shall maintain the following panels: (1) a panel of doctors designated by the Medical Society of New Jersey; (2) a panel of doctors designated by the New Jersey Society of Osteopathic Physicians and Surgeons; and (3) a panel of attorneys with trial experience designated by the Supreme Court. Additions or deletions may be made to these panels at any time at the discretion of the Administrative Director of the Courts.

Note: Adopted July 24, 1978 to be effective September 11, 1978.

4:21-4. Appointment of Panel; Notification and Disqualification

(a) **Appointment of Panel.** All hearings shall be held before a panel consisting of a judge who shall preside as chairman, a doctor, and an attorney, all of whom shall be appointed in accordance with the following provisions.

(1) The chairman shall be the same judge to whom the action has been assigned pursuant to R. 4:21-2(b) unless the Assignment Judge, in extraordinary circumstances and for good cause shown, otherwise orders.

(2) The doctor shall be selected by the Administrative Director of the Courts from the medical or osteopathic panel, as appropriate, depending upon the discipline of the defendant doctor or doctors. Where practicable, the doctor panelist shall be a practitioner in the field of speciality involved in the case.

(3) The attorney shall be selected by the Administrative Director of the Courts.

(b) **Notification and Disqualification.** The Administrative Director of the Courts shall notify the doctor and attorney that they have been selected to be members of a panel for a particular case and shall request that they disclose any circumstances likely to create a presumption of bias or which they believe might otherwise disqualify them. Information disclosed in response to such a request shall then be forwarded to all parties together with notification to all parties of the identity of the panel members. Within fifteen days after mailing of notification of the panel selection to all parties, any party may file and serve written objection to the designation of the doctor or attorney, with supporting reasons therefor. This objection shall be heard by the judge presiding as chairman of the panel. Copies of the written objection and the judicial determination thereon shall be sent to the Administrative Director of the Courts. Whenever a panelist is disqualified, the Administrative Director of the Courts shall select a substitute in accordance with the provisions herein.

(c) **Time for Hearing.** When an acceptable panel is constituted, the presiding judge shall fix the time and place for hearing. All applications for adjournment shall be made to the presiding judge.

Note: Adopted July 24, 1978 to be effective September 11, 1978.

4:21-5. Hearing Before Panel; Findings; Effect

(a) **Participation at Hearing; Confidentiality.** The hearing shall be informal and without a stenographic record. Except as otherwise provided in this section, no statement or expression of opinion made in the course of the hearing shall be admissible in evidence either as an admission or otherwise in any trial of the action. If any of the parties to the hearing fails to appear or to comply with the rules set forth herein respecting the hearing, the presiding judge may enter such order as justice requires.

(b) **Presentation of Facts.** The parties shall set forth their factual and legal contentions in any of the following forms: narrative statement, question and answer, written statement, sworn testimony, or in such other form as the panel deems appropriate. On application by any party, the presiding judge may issue subpoenas and compel compliance therewith in accordance with the appropriate provisions of Rule 1:9 (Subpoenas). All witnesses and parties called to testify may be cross-examined by any adverse party, subject to the discretion of the presiding judge. The parties shall submit all written material including the original or copies of all hospital records and other reports upon which they shall rely to the members of the panel no later than ten (10) days prior to the hearing by filing same with the presiding judge.

(c) **Scope of Panel Jurisdiction.** The parties to the hearing may, at any time, enter into stipulations concerning procedure and evidence and may agree to extend the jurisdiction of the panel to determine damages. Co-defendants in the action who are not doctors may submit to such hearing and thereby become subject to the same rights and sanctions affecting the plaintiff and defendant doctor or doctors.

(d) **Expert Witness.** The panel may request an additional doctor having particular expertise in the speciality involved to assist it in the determination of the claim. Such doctor shall make a report to the panel. The panel shall determine the fee and expenses to be paid to such doctor and the parties to the hearing shall share equally in such fee and expense. This doctor and the doctor member of the panel may be called at subsequent trial of the action as a witness by any of the parties. The party calling such witness at trial shall pay all reasonable fees and expenses of the doctor.

(e) **Findings; Admissability at Trial.** At the conclusion of the hearing, the panel shall make specific findings of fact as to each medical issue and an appropriate order determining whether the claim is based on reasonable medical probability shall be served on all parties

to the action. This order shall be filed and sealed in accordance with R. 4:21-7 with full restrictions as to all parties and their attorneys against disclosure. If the order setting forth the panel's disposition is unanimous, the findings contained therein shall be admissible in evidence at trial upon the request of any party to the hearing. The recommendation shall not be binding upon the jury or upon a judge sitting as the trier of the facts, but shall be accorded such weight as the jury or the judge sitting as the trier of the facts chooses to ascribe to it in view of all the relevant evidence adduced at trial.

(f) **Documents; Identification; Disposition.** All writings as defined in Evidence Rule 1(13) upon which the parties rely at the hearing shall be marked for identification and shall be returned to the respective parties after entry of the order.

(g) **Participation of Panel Members in Trial.** The judge presiding at the hearing shall not preside at the trial, nor shall any panel member participate in the trial either as counsel or witness except as otherwise provided herein.

Note: Adopted July 24, 1978 to be effective September 11, 1978.

4:21-6. Rehearing

The panel by majority vote of its members may grant a single rehearing, but such rehearing shall not be granted solely because the plaintiff or defendant doctor or doctors has substituted counsel.

Note: Adopted July 24, 1978 to be effective September 11, 1978

4:21-7. Proceedings to be in Camera; Secrecy

All proceedings, records, findings and recommendations of the panel shall be confidential and shall not be used in any proceeding other than the trial of the action. All proceedings shall be in camera and no records shall be made thereof.

Note: Adopted July 24, 1978 to be effective September 11, 1978.

Report from the Foundation

Daniel J. O'Regan, M.D.
Medical Director

Janus, the Roman god for whom this month was named, had the ability to look ahead and backward at the same time. The physician of the late 1970s needs to look in all directions, not just two. The last quarter of the old year can be characterized by inflationary trends, balance of payment deficits, and further declines in the value of the dollar. The "conservative" posture predicted for those elected to Congress is said to mean resistance to new Federal spending. While this will deter enactment of National Health Insurance, it will mean further pressure to reduce the costs of health care. While the voluntary effort is bearing fruit, Mr. Califano insists that the mandatory "cap" bill must be reintroduced. Champions of the Health Planning law promise more stringent activities. The Secretary of HEW has directed the attention of the National Health Planning Council toward developing "productivity standards" for health care services of all kinds, including physicians' activities. The Federal Trade Commission appears to be on a crusade to destroy ethical standards which were developed by organized medicine for the protection of the public. Onslaughts from "health care practitioners other than physicians," with the help of the courts, threaten to disrupt relationships between specialty societies and the AMA. The daily recitation that the Carter Administration is "opposed" to mandatory wage and price controls should be taken with some salt. The assaults appear to come from all directions. NJFHCE will help to keep you aware of them during 1979 (1984 is only five years hence!).

CMDNJ Notes*

Stanley S. Bergen, Jr., M.D.
President

With the approval of an $8.7 million contract by the U.S. Department of Health, Education, and Welfare for a Camden Area Health Education Center (AHEC) program, the unique and long-awaited project in health care and education for southern New Jersey has moved into its formal planning stage.

An acting director has been named to coordinate plans for the AHEC project. The first phase, slated to last one year, will focus on the development of details such as formal agreements between CMDNJ and the community hospitals participating in the program, the appointment of a Program Advisory Committee, and the establishment of the program's organizational and management structure. A second phase, also to last one year, is designated for development, and the final four-year stage will be devoted to implementation.

The initiation of the AHEC program is significant to all health professionals in New Jersey. The program's major goals are to fill the gaps in Camden's health system, increasing the accessibility of high-quality care in that underserved area, and to create a center for health professionals' education at all levels in the city which will serve all of South Jersey.

What is unique in the Camden program is that it marks the consolidation of resources—local, state, and federal—and a joining together of professionals from the entire spectrum of health professions in a common effort to achieve a common goal.

The AHEC program will build upon the foundation of the community's existing health care resources. Four outpatient facilities have been designated as AHEC sites: Cooper Medical Center, Our Lady of Lourdes Hospital, West Jersey Hospital-Northern Division, and the facilities of the Camden County Health Department. In addition, educational programs will be based at the AHECs as well as at other local health care and educational facilities.

The State's contribution will be made through CMDNJ. The program provides a framework through which to channel the College's vast resources in health education into South Jersey. Four CMDNJ units will be participating in the AHEC project: the CMDNJ-New Jersey School of Osteopathic Medicine, the CMDNJ-New Jersey Dental School, the CMDNJ-School of Allied Health Professions, and the Office of Consumer Health Education of CMDNJ-Rutgers Medical School. The College's goal will be to establish in South Jersey a network of health professions' education programs that will at once assist in serving the area's immediate health care needs and assist in creating an adequate pool of health manpower for the future.

The educational component of the AHEC program will cover the entire spectrum of health from patient and consumer education to continuing education for practicing professionals.

The undergraduate program of the CMDNJ-New Jersey School of Osteopathic Medicine already is based in South Jersey for its clinical training. Since osteopathy is strongly oriented toward primary care, the school will have close ties to the AHECs.

In addition, CMDNJ plans to foster additional postgraduate training pro-

grams, particularly in the area of primary care, and to offer continuing education programs designed to help professionals refresh their knowledge and update their skills.

Through the CMDNJ-School of Allied Health Professions, the College will be working toward the establishment of a network of training programs geared to meet the needs of South Jersey. New programs will be established, and existing programs expanded, in an effort to provide area professionals with the support personnel they need. The school will work with local colleges and universities as well as health care groups in developing programs in fields such as nurse midwifery, physician assisting, and emergency medical technology.

The College also will establish and expand programs in patient education at community facilities, to underline the need for preventive medicine and the patient's role in his own health care. These programs will focus on topics such as nutrition, diabetes, heart attack prevention, and child care. By introducing a broad range of programs in health education in Camden, we hope to promote an improved academic atmosphere for all health professionals.

The primary goal of the AHEC program is to turn around the total health care picture in Camden. Southern New Jersey, and Camden particularly, long have suffered from a shortage of health professionals and resources. Camden, in fact, has been designated by the Federal government as a critical manpower shortage area for physicians. The city has the worst infant mortality rate in New Jersey, with an infant death rate of 37.6 percent. In a city of more than 100,000 residents, there are only .542 practicing primary care physicians per 1,000 people.

The presence of CMDNJ-New Jersey Medical School in Newark has done much to improve the health picture in that city during the past ten years. We hope that the presence of AHEC in Camden will have the same impact over the next ten years.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Office of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ALLERGY/IMMUNOLOGY—Alan R. Green, M.D., 1315 Ethel Street, Glendale, CA 91207. New York Medical 1971. Partner, associate in single-specialty group, or purchase of practice. Available August 1979.

ANESTHESIOLOGY—Parvin Javadi, M.D., 57 Montague Street, Apt. 9G, Brooklyn, New York 11201. Shiraz (Iran) 1964. Board eligible. Group, solo, partnership. Available.

CARDIOLOGY—Dhirendra Mohan, M.D., 757 Main Street, Apt. 30, South Portland, ME 04106. King George (India) 1967. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

Micha Oren, M.D., Beth Israel Hospital/Harvard Medical School, Division of Cardiology, 330 Brookline Ave., Boston, MA 02213. Tel Aviv (Israel) 1972. Subspecialty, internal medicine. Board certified (IM). Group, partnership, solo, hospital-based. Available July 1979.

Michael M. Neumann, M.D., 5415 North Sheridan Road, Chicago, IL 60640. Dusseldorf (Germany) 1971. Subspecialty, internal medicine. Board certified (IM). Group or institution. Available.

Dominick J. Passalacqua, M.D., 2392 East 5th Street, Brooklyn, New York 11223. Bologna (Italy) 1972. Cardiac laboratory for invasive and non-invasive procedures. Available January 1979.

Michael J. Zema, M.D., 23 Rosemont Avenue, Elmwood Park, New Jersey 07407. Cornell 1974. Board certified—IM. Board eligible—Cardiology. Group, partnership, solo (with or without part-time institution). Available July 1979.

Muljibhai J. Thakkar, M.D., 255-25 75th Avenue, Glen Oaks, New York 11004. Baroda (India) 1972. Solo. Available July 1979.

FAMILY PRACTICE—Jack A. Devor, M.D., 828 Cape View Drive, Fort Myers, Florida 33907. Hahnemann 1966. College or university health service. Available.

Elmer S. Gilo, M.D., St. Joseph Hospital, Stamford, CT 06904. University of the East (Philippines) 1973. Board eligible. Group, solo, partnership; also teaching. Available July 1979.

Charles A. Joseph, M.D., 99 Morris Avenue, Summit 07901. CMDNJ 1976. Board eligible. Group or partnership. Available July 1979.

HEMATOLOGY—Jagmohan K. Kalra, M.D., 2 Robin Lane, Plainview, N.Y. 11803. Lady Hardinge (India) 1970. Subspecialty, oncology. Board certified (IM) (Hematology). Group, partnership, hospital. Available July 1979.

INTERNAL MEDICINE—Harshad R. Mehta, M.D., 1007 Elkin Residence, 5501 North 11th Street, Philadelphia, PA 19141. T.N.M. College (India). Subspecialty, cardiology. Board certified. Group, partnership, solo, institutional. Available July 1979.

Jon David Green, M.D., 509 Fairview Avenue, Orange 07050 (Apt. C-1). CMDNJ 1974. Subspecialty, gastroenterology. Board certified. Solo, partnership, group. Available July 1979.

Richard M. Nisman, M.D., 550 Rossmore Road, Richmond, VA 23225. Medical College of Virginia. Subspecialty, gastroenterology. Board certified. Group, partnership, solo. Available January 1979.

Peter K. W. Chak, M.D., 200 Bethel Loop, Apt. 20D, Starrett City, Brooklyn, New York 11239. Hong Kong 1970. Board eligible. Group, partnership, associate, solo. Available June 1979.

Robert S. Goldblatt, M.D., 150 Division Street, Derby, Connecticut 06418. George Washington 1974. Subspecialty, gastroenterology. Board eligible. Group, partnership. Available July 1979.

Alan M. Nelson, M.D., 41B Monroe Street, Milford, Connecticut 06460. Georgetown 1974. Subspecialty, gastroenterology. Board certified (IM). Group, partnership. Available July 1979.

Vithal Kusuma, M.D., 1227 South Harlem, #504, Berwyn, Illinois 60402. Osmania (India) 1970. Subspecialty, gastroenterology. Board certified (IM). Hospital, group, solo. Available July 1979.

Steven Nussbaum, M.D., 891 Clopper Rd., Apt. B-1, Gaithersburg, Maryland 20760. SUNY—Downstate, 1974. Subspecialty, gastroenterology. Board certified (IM). Group, partnership. Available July 1979.

Stephen A. Atlas, M.D., 159 Hawthorne Avenue, Central Islip, NY 11722. Wisconsin. Board eligible. Group or partnership. Available July 1979.

Yugal K. Maheshwari, M.D., 10555 Fondren, Apt. #727, Houston, Texas. J. N. Medical College (India) 1970. Subspecialty, endocrinology. Board eligible. Partnership, associate, single or multi-specialty group. Available July 1979.

Michael D. Burday, M.D., 139-1 Kirkbride Road, Voorhees 08043. Georgetown 1976. Group or partnership. Available July 1979.

Ravi K. Malpani, M.D., 1165 Route 22, Apt. 22, North Plainfield 07061. Osmania (India) 1972. Subspecialty, pulmonary medicine. Board eligible. Group, solo, partnership, or hospital-based. Available July 1979.

John Kassabian, M.D., 25 Burnett Terrace, West Orange 07052. CMDNJ 1974. Sub-

specialty, pulmonary medicine. Board certified. Any type practice. Available July 1979.

OPHTHALMOLOGY—Pradyumna C. Butala, M.D., 2520 S. King Drive, Apt. 505, Chicago, Illinois 60616. Baroda (India) 1968. Board eligible. Partnership, associate (fulltime). Available January 1979.

Zdenka F. Macek, M.D., 271 Nandina Terrace, Philadelphia 19116. Charles University (Czechoslovakia) 1966. Board eligible. Group, partnership, association, institution. Available.

PEDIATRICS—Narendra V. Ambani, M.D., 1945 Corlies Avenue, Neptune 07753. Baroda (India) 1973. Board eligible. Solo, partnership, or group. Available July 1979.

R. P. Vasani, M.D., 1575 Boston Ave., #C-12. Bridgeport, Connecticut 06610. M.S. University (India) 1974. Board eligible. Group or partnership. July 1979, or sooner.

Mehboob A. Sachani, M.D., 1 Slate Creek Rd., Apt.-3, Cheektowaga, New York 14227. Seth G. S. Medical (India) 1974. Board eligible. Any type of practice. Available July 1979.

C. V. R. N. Reddy, M.D., 57 Rupert Ave., Staten Island, NY 10314. Kakatiya (India) 1968. Board eligible. Group, partnership, solo. Available.

Theodore Sher, M.D., 170 West End Avenue, New York, NY 10023. SUNY—Downstate 1974. Special interest, pediatric allergy. Board eligible. Group or partnership. Available July 1979.

Warren P. Silberstein, M.D., 17 Bartlett Drive, Middletown, NY 10940. SUNY—Downstate. Board eligible. Group practice. Available July 1979.

Vijaya Lakshmi Malpani, M.D., 1165 Route 22, Apt. 22, North Plainfield 07061. Osmania (India) 1972. Board eligible. Group, solo, hospital-based, partnership. Available July 1979.

PSYCHIATRY—Elaine Nicola, M.D., 117 Grove Street, Ramsey 07446. Northwestern 1970. Board certified. Group, partnership, or clinic. Available February 1979.

PULMONARY DISEASES—Peter Y. Lee, M.D., 319 East 24th Street, Apt. 21D, New York 10010. NYU 1974. Board certified (IM). Board eligible (pulmonary diseases). Group, institution, association. Available July 1979.

Shreedhar B. Shetty, M.D., 116B Davey Street, Bloomfield 07003. Kasturbha (India) 1968. Board eligible. Group. Available July 1979.

SURGERY, GENERAL—Charles P. Clericuzio, M.D., 13030 Trail Hollow, Houston, Texas 77079. Wisconsin 1971. Subspecialty, colonoscopy. Board certified. Group, hospital-based, or academic. Available.

Hank S. Lee, M.D., Route 4, Haleyville, Alabama 35565. Catholic Medical College, 1966. Board certified. Solo or partnership. Available.

Ivan A. Shulman, M.D., 785 Burnett Avenue, Apt. 7, San Francisco, CA 94131. Pittsburgh 1972. Board eligible. Group or partnership. Available July 1979.

P. Thaker, M.D., 5 Centennial Drive, Norwood, MA 02062. Baroda (India). Board eligible. Any type practice. Available.

Michael J. Kassouf, M.D., 4 Dartmouth Ave., Apt. 3A, Bridgewater, New Jersey 08807. French Faculty (Beirut, Lebanon) 1970. Board eligible. Group, partnership, solo. Available September 1979.

William A. Schuckardt, Jr., M.D., 13500 Shaker Road, Cleveland, Ohio 44120. Case Western Reserve 1974. Board eligible. Group, solo, or partnership. Available July 1979.

John L. DeBoer, M.D., 1242 B Cedar Street, Fort Dix. Vermont 1971. Board certified. Single-specialty group, partnership, multi-specialty group, solo. Available June 1979.

David W. Fricke, M.D., 1020 Orr's Bridge Road, Mechanicsburg, PA 17055. University of Pennsylvania 1960. Board certified. Any type, partnership or group practice preferred. Available.

M. Y. Dhar, M.D., 318 East 15th Street, Apt. 8-M, New York, NY 10003. G. R. Medical College (India). Board eligible. Any type practice. Available July 1979.

Stuart A. Engel, M.D., 545 39th Avenue, San Francisco, CA 94121. Johns Hopkins 1974. Group or partnership. Available August 1979

Michael F. Lane, M.D., 7901 Henry Avenue, Apt. C-411, Philadelphia, PA 19128. SUNY—Downstate 1973. Board eligible. Group or partnership. Available July 1979.

SURGERY, ORTHOPEDIC—Ronald Scheinzeit, M.D., 3432 Alpine Court, Lexington, Kentucky 40502. Albany 1974. Board eligible. Group, association, partnership. Available July 1979.

Ronald B. Greene, M.D., 1405 Bay Drive, Miami Beach, FL 33141. Temple 1972. Board eligible. Solo, group, or association. Available July 1979.

SURGERY, THORACIC—Chukuma I. Okadigwe, M.D., 590 Flatbush Avenue, Apt. 14-P, Brooklyn, NY 11225. Colorado 1968. Board eligible. Group, partnership, solo. Available.

UROLOGY—Howard B. Somers, M.D., 1153 East 72nd Street, Brooklyn, New York 11234. Guadalajara 1973. Board eligible. Partnership, group, institution. Available July 1979.

Arnold M. Grebler, M.D., 19 Spartan Avenue, Staten Island, NY 10202. Bologna (Italy) 1974. Partnership or group. Available July 1979.

Stewart L. Feldman, M.D., 34 Hunter Circle, Mount Holly 08060. Vermont 1971. Board eligible (general surgery). Group, partnership. Available July 1979.

Ekiong C. Tan, M.D., 8478 Ohern Street, Omaha, Nebraska 68127. Santo Tomas (Philippines) 1970. Board eligible. Group or partnership. Available July 1979.

Daniel H. Piazza, M.D., 741 Peninsula Court, Ann Arbor, Michigan 48105. Illinois 1972. Board eligible. Partnership, single or multi-specialty group. Available July 1979.

Gerald A. Goldman, M.D., 2913 Willowwood Drive, Erie, PA 16500. Guadalajara 1972. Board eligible. Association leading to partnership. Available July 1979.

213th Annual Meeting
May 12-15

Headquarters Hotels

Holiday Inn—Boardwalk Regency

Atlantic City

(See housing application—p. 55, this issue)

LETTER TO THE JOURNAL

CMDNJ Physical Therapy Program

November 8, 1978

Dear Doctor Krosnick:

This is in reference to the College of Medicine and Dentistry of New Jersey (CMDNJ) Notes, by Stanley S. Bergen, M.D., appearing in *The Journal,* November 1978, page 868.

The College is to be congratulated on the institution of the Physical Therapy program, "to ease the shortage of health manpower in New Jersey, . . ." Up until now, New Jersey residents have had to seek this training outside New Jersey, and many schools would not accept out-of-state students. We have had a shortage of physical therapists in New Jersey, a condition which will be ameliorated by this program. It is to be hoped that classes in the future will be large, since the demographies of an aging population are likely to require even greater numbers of qualified physical therapists.

A small cavil—the characterization of the physical therapist's work with "orthopedically impaired patients"—I think that does not adequately define the scope of physical therapy. As an example, the patient with hemiplegia following a stroke is excluded from the definition. Here, the patient with stroke is the largest number by diagnosis treated per year, and that number is increasing. It also excludes large numbers of patients with arthritis not treated by orthopedic surgery whose lives are enhanced with proper regimens including physical therapy, as well as other non-orthopedic problems.

I thank you for the opportunity to add to Doctor Bergen's announcement.

(signed) Percy L. Miller, M.D.
Director, Physical Medicine
and Rehabilitation
Hospital Center at Orange

PERSONAL ITEMS

Dr. Saffron Honored

The following are the remarks of Morris H. Saffron, M.D., on the occasion of the dedication of the Academy of Medicine's Rare Book Collection, presented in Dr. Saffron's name to the CMDNJ—George F. Smith Library on October 18, 1978. The inscription on the plaque reads: "The Academy of Medicine of New Jersey presented its Rare Book Collection to the CMDNJ—George F. Smith Library of the Health Sciences, to be housed in the History of Medicine Room in honor of Morris H. Saffron, M.D., Ph.D., Scholar and Bibliophile, 1978."

"President Bergen, Vice-President Vevier, Dean Lanzoni, Ladies and Gentlemen: I have jotted down these lines because I anticipated—and quite correctly—that in the emotional stress of this occasion I might well find myself in the unusual yet nonetheless embarrassing position of being at a loss for words. I need not say that I feel this to be one of the most momentous days in my life, and that no other type of celebration could conceivably have been more meaningful to me, or have given me more reason to rejoice. From my father who was a great scholar I early imbibed a reverence for the printed book which has persisted to this day, and the great joy I have had in collecting books for well over half a century has been equalled only by the pleasure—sometimes I admit mixed with a tinge of poignancy—of seeing them depart forever from my shelves to serve a wider audience. Although Columbia, my alma mater, has been the principal beneficiary of these gifts, I might mention that at least five other institutions preserve today some of my former treasures.

"The thought that this fine collection —one which I have nurtured and protected ever since I became chairman of the Academy library committee some thirty-odd years ago—will now perpetuate my name is at once awesome and deeply moving, and inspires in me a sense of kinship with the noble authors represented here who have contributed so greatly to humanity, and whose very presence seems to radiate among us today. I could go on in this vein, but obviously the depth of my feeling and the sincerity of my gratitude is by now apparent to all of you. I would be remiss indeed were I not to voice my thanks to Drs. Leo Siegel and Victor Parsonnet, both former presidents of the Academy, for the concept which led to today's events, and to Philip Rosenstein and Charles Heitzmann and their respective staffs for so tastefully carrying out the prepared plans. May I also acknowledge here the presence of my small family and a few intimate friends who have joined me on this happy occasion. Among other colleagues I am happy to see Gertrude

Annan, librarian emeritus of the New York Academy of Medicine, without whose help much of my literary production might have withered on the vine; Dr. Genevieve Miller, director of the Dittrick Museum of Historical Medicine of Cleveland and currently president of the American Association for the History of Medicine; Dr. Clifford Lord, whom I first met as president of Hofstra University, and who is now the director of the New Jersey Historical Society; Dr. James Rogers, currently president of the Academy and a former president of the Medical Society of New Jersey; and Dr. William D. Sharpe, noted scholar, now editor of the *Bulletin of the New York Academy of Medicine.*

"I should like to conclude these brief but heartfelt remarks with two Latin mottoes which seem appropriate, as they relate to my personal credo and the significance of the present gathering. The first comes from Jean Grolier, sixteenth century book collector and patron saint of the famous club which bears his name and whose president, Robert Graff, is with us today. Grolier had embossed on all the bindings of his cherished library the words *Ioannis Grolieri et amicorum,* implying of course that he considered himself to be merely a temporary custodian of his precious books, and that they belonged not only to him but to his entire circle of friends and by extension to all mankind; the other states quite simply *amor librorum nos unit,* which I take to mean that the love of books creates among its votaries an everlasting bond. I should like to carry away with me the belief that no meeting could more aptly manifest the truth of that admirable sentiment than the one we are participating in this very afternoon. Again I thank you from the bottom of my heart."

Dr. Nichols Elected to National Cancer Institute Advisory Board

Dr. Warren W. Nichols of Cherry Hill, a member of the Camden County Medical Society, has been elected to the Scientific Advisory Board, Cancer Cause and Prevention Council of the National Cancer Institute, Bethesda, Maryland. Dr. Nichols is Assistant Director of the Institute for Medical Research, Camden, where he was awarded the S. Emlen Stokes Professor of Genetics chair in 1975. He also serves as head of the Department of Cytogenetics at the Institute, a research facility noted for its studies in the fields of cancer and genetics.

Dr. Nichols earned his medical degree from Jefferson Medical College of Philadelphia, and a Fil. Lie degree in genetics and Ph.D. in cytogenetics from the University of Lund, Sweden.

He has served as a consultant to the Secretary's Commission on Pesticides and Their Relationship to Environmental Health for the U.S. Department of Health, Education, and Welfare, as a permanent panel member for the United States-Japan Cooperative Medical Science Program, and as Co-Chairperson for the US-USSR Program on Mammalian Somatic Cell Genetics Related to Neoplasia for the National Cancer Institute.

Dr. Nichols was Secretary of the Environmental Mutagen Society and served as a Councilor to that society. He also holds professorships in human genetics and pediatrics at the University of Pennsylvania School of Medicine.

Dr. Schauer Named Vice-President, AAFP

Edward A. Schauer, M.D., of Farmingdale, Monmouth County, was named Vice-President of the American Academy of Family Physicians at the September 25, 1978 election held by the Congress of Delegates of the Academy in San Francisco. Dr. Schauer, a diplomate of the American Board of Family Practice, and a member of the attending staff and Director of the Department of Family Practice at Jersey Shore Medical Center in Neptune, is an active member of MSNJ, having served three terms as member and vice-chairman of the Council on Mental Health, several years as a member of the Committee on Medicare Peer Review, and long as a representative from Monmouth County to our House of Delegates.

213th ANNUAL MEETING, MSNJ

COFFEE LOUNGE

Boardwalk Regency

Sponsored by **THE PRUDENTIAL INSURANCE COMPANY OF AMERICA**

Representatives will be available to answer questions on Medicare and Medicaid

WEEKLY CARDIOLOGY CONFERENCES

(Approved for 2 hours Cot. I Credit for C.M.E.)

Objective of Program: Updote of diognostic methods in cordiology; indicotions ond results of cordioc surgery.

PROGRAM CHAIRMAN: Horry Goldberg, M.D., Professor of Medicine
Temple University School of Medicine
Chief of Cordiology, Deboroh Heort ond Lung Center

With speciolists on Deboroh's stoff presenting Echocordiogroms, ECG's, Treodmill testing, Vector-Cordiogroms, Nucleor Scons, Coronory Cines ond Ventriculogroms, Cordioc Coth. Doto, Angiogroms ond Surgicol Methods.

Guest Lecturers: A distinguished guest lecturer will present speciol topics on Cordiology once o month.

THURSDAYS (Every Week) 2:15 P.M.—4:00 P.M. (September 7, 1978 to June 28, 1979)

LOCATION: DEBORAH HEART AND LUNG CENTER,
BROWNS MILLS
(1st. Floor Conf. Room)

GASTROENTEROLOGY for the Primary Care Physician

will be presented by the
Division of Gastroenterology
and the
Program of Continuing Education
of the
University of Maryland
School of Medicine
on
March 9th & 10th, 1979
at the
Cross Keys Inn, Columbia, Maryland

Designed to focus on problems of the practicing physician in the primary care setting, the program will cover aspects of nutrition, peptic ulcer disease, GI endoscopy, hepatitis, liver disease, surgical management of morbid obesity and GI disease in the elderly.

For more information contact: The Program of Continuing Education, 10 South Pine Street, Baltimore 21201 or phone (301) 528-3956.

CME CALENDAR

This listing is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, the Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the College of Medicine and Dentistry of New Jersey. For information on accreditation, please contact the sponsoring organization(s), indicated by italics – last line of each item.

ANESTHESIOLOGY

February

24 Anesthesiology Presentations
9 a.m.-1:30 p.m.—St. Barnabas Medical Center, Livingston
(Educational Council for Anesthesiology of New Jersey and AMNJ)

March

24-25 20th Annual Postgraduate Seminar
9 a.m.-5 p.m., Sat.; 9 a.m.-1 p.m., Sun.—Hyatt House, Cherry Hill
(New Jersey State Society of Anesthesiologists and AMNJ)

MEDICINE (includes Family, Internal, and General Medicine and Dermatology)

February

1 Cardiology Conferences
8 2:15-4:15 p.m.—Deborah Heart and
15 Lung Center, Browns Mills
22 *(Deborah Heart and Lung Center and AMNJ)*

1 Virology
8 4-6 p.m.—Institute for Medical
15 Research, Copewood St., Camden
22 *(Institute for Medical Research and AMNJ)*

2 Antiarrhythmic Medications
9 Hypertension Update
16 Radiology of the Heart—X-ray, Scan, and Echo
23 Differential Diagnosis of Heartblocks
11:45-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Medical Center)

7 Medical Grand Rounds
11:30 a.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

7 Dinner Meeting
6:30-9:30 p.m.—Holiday Inn, East Orange
(AMNJ Endocrinology Section)

7 Childhood Leukemia and Solid Tumors
14 Ulcer Disease
21 Inflammatory Bowel Disease
28 Ischemic Brain Syndromes
9-11 a.m.—Riverview Hospital, Red Bank
(Riverview Hospital and AMNJ)

7 Environmental Hazards
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's, Denville and AMNJ)

7 Obesity
11:30 a.m.-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

7 Advances in Medicine
14 9:30 a.m.-11 a.m.—Bergen Pines County
21 Hospital, Paramus
28 *(Bergen Pines County Hospital and AMNJ)*

7 Infertility
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

7 Nephrology: Fluid and Electrolyte Imbalance
14 Physiological Parameters in Critical Care Therapy
21 Rehabilitation Surgery
28 Echocardiography
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ, and AAFP)

7 Endocrine Conferences
14 3:30-5 p.m.—Rotates between Newark
21 Beth Israel Medical Center, Martland
28 Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

7 Urinary Tract Infections
14 Blood Pressure and the Kidney
21 Diuresis and Antidiuresis
28 Diagnosis and Overdiagnosis of Digestive Disease
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ, and AAFP)

13 Clinical Immunology
20 Sepsis Shock
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

13 Acute and Chronic Hepatitis
8 p.m.—Paul Kimball Hospital, Lakewood
(Paul Kimball Hospital and AMNJ)

13 Allergy and Immunology
8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

13 Bullous Diseases
8-10 p.m.—Schering Corporation, Kenilworth
(NJ Dermatological Society and AMNJ)

13 Cardiac Rehabilitation
12:45-2 p.m.—Helene Fuld Medical Center, Trenton
(American Heart Association, Mercer County Chapter)

14 Blood Gases
1:30-2:30 p.m.—John E. Runnells Hospital, Berkeley Heights
(John E. Runnells Hospital and AMNJ)

15 Impact of Genetics on Medical Practice
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

15 Regional Chest Conferences
7:30-9:30 p.m.—St. Michael's Medical Center, Newark
(New Jersey Thoracic Society and AMNJ)

20 Use and Abuse of Antibiotics
8 p.m.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

20 Infectious Diseases—Endotoxic Shock
7-8 p.m.—Irvington General Hospital
(Irvington General Hospital and AMNJ)

20 Breast Cancer
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

28 Cardiac Grand Rounds
3-4:30 p.m.—CMDNJ-NJ Medical School
(American Heart Association and AMNJ)

March

1 Cardiology Conferences
8 2:15-4:15 p.m.—Deborah Heart and
15 Lung Center, Browns Mills
22 *(Deborah Heart and Lung Center and*
29 *AMNJ)*

2 Lymphomas
9 Carcinoma of the Colon
14 CA of the Cervix
16 CA of the Urinary Tract
23 Chemotherapy Review
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

6 Hypertension in Cardio-Vascular Renal Disease
13 Use of Psychtrophic Drugs in Cardiac Patients
8-9 a.m.—Greater Paterson General Hospital
(Greater Paterson General Hospital and AMNJ)

7 Dinner Meeting
6:30-9:30 p.m.—Holiday Inn, East Orange
(AMNJ Endocrinology Section)

7 Medical Grand Rounds
11:30 a.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital,

East Orange
(AMNJ Endocrinology Section)

7 **Human Immmnologic Response in Lymphocyte Function**
14 **Nuclear Cardiology**
21 **Infections Diseases**
9-11 a.m.—Riverview Hospital, Red Bank
(Riverview Hospital and AMNJ)

7 **Gall Bladder Disease**
14 **Diabetic Retinopathy and Retinal Neo-Vascularization**
28 **Renal Failure**
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

7 **Advances in Medicine**
14 9:30 a.m.-11 a.m.—Bergen Pines County
21 Hospital, Paramus
28 *(Bergen Pines County Hospital and AMNJ)*

14 **Diabetes**
21 **Orthotics and Prosthetics**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ and AAFP)

7 **Endocrine Conferences**
14 3:30-5 p.m.—Rotates between Newark
21 Beth Israel Medical Center, Martland
28 Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

7 **Vasodilator Therapy in Heart Disease**
14 **Current Problems in Transplantation**
21 **Common Gastrointestinal Disorders Among Children**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

8 **Virology**
15 4-6 p.m.—Institute for Medical
22 Research, Copewood St., Camden
29 *(Institute for Medical Research and AMNJ)*

10 **Vascular Injuries**
1-5 p.m.—Newark Beth Israel Medical Center
(N.J. Chapter, American Trauma Society, Vascular Society of N.J. and AMNJ)

13 **Immunology**
8-10 p.m.—Schering Corporation, Kenilworth
(NJ Dermatological Society and AMNJ)

13 **Acute Renal Failure**
20 **Chronic Renal Failure**
27 **Fluid and Electrolyte Imbalance**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

13 **Nutrition and Aging**
8 p.m.—Paul Kimball Hospital, Lakewood
(Paul Kimball Hospital and AMNJ)

14 **Medical Lecture Series**
21 1—3 p.m.—Christ Hospital, Jersey City
28 *(Christ Hospital and AMNJ)*

15 **Treatment of Bronchospasm**
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

15 **Regional Chest Conferences**
7:30-9:30 p.m.—Overlook Hospital, Summit
(N.J. Thoracic Society and AMNJ)

20 **Gastroenterology—Proper Use of Endoscopy**
8:30-9:30 p.m.—Omar's Restaurant, Saddle River Rd., Fair Lawn
(Fair Lawn Memorial Hospital and AMNJ)

20 **Nuclear Medicine**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

27 **Head and Neck Cancer**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

28 **Cardiac Grand Rounds**
3-4:30 p.m.—NJ Medical School, Newark
(American Heart Association, NJ Affiliate and AMNJ)

NEUROLOGY/PSYCHIATRY

February

1 **Psychotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, South Orange
(Group for Advanced Psychiatric Study and AMNJ)

1 **Psychiatric Lecture Series**
8 11 a.m.-12 noon—Greystone Park
15 Psychiatric Hospital
22 *(Greystone Park Psychiatric Hospital and AMNJ)*

5 **Intermittent Treatment of a Schizophrenic**
8-10 p.m.—39 Crescent Ave., Passaic
(Essex Psychiatric Seminar and AMNJ)

5 **Neuroscience Conferences**
12 11:30 a.m.-12:30 p.m.—Bergen Pines
19 County Hospital, Paramus
26 *(Bergen Pines County Hospital and AMNJ)*

6 **Headache**
8-9 p.m.—Burdette Tomlin Memorial Hospital, Cape May Court House
(Burdette Tomlin Memorial Hospital and AMNJ)

6 **Seminar on Law and Psychiatry**
13 3:30-5:30 p.m.—Rutgers Law School,
20 Newark
27 *(Rutgers University Law School and AMNJ)*

6 **Introduction to Family Therapy**
13 7:30-9:30 a.m.—Trenton Psychiatric
20 Hospital
27 *(Trenton Psychiatric Hospital and AMNJ)*

7 **Current Issues in Psychophysiologic Disorders**
7:30-9:30 p.m.—Mayfair Farms, West Orange
(Tri-County Chapter, N.J. Psychiatric Association and AMNJ)

7 **Clinical Pathology Grand Rounds**
14 12 noon-1 p.m.—NJ Medical School,
21 Newark
28 *(CMDNJ and AMNJ)*

7 **Endocrine Conferences**
14 3:30-5 p.m.—Rotates between Newark
21 Beth Israel Medical Center, Martland
28 Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

7 **Depression**
21 Title to be Announced
1:30-3 p.m.—NJ Medical School, Newark
(NJ Medical School and AMNJ)

7 **Psychosomatic Medicine**
14 **Hospital Psychiatry—Principles**
21 **Hospital Psychiatry—Treatment**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

8 **Voice of Conscience**
8:30-10:30 p.m.—Location to be announced
(NJ Psychoanalytic Society and AMNJ)

13 **Clinical Immunology**
20 **Sepsis Shock**
27 **Laboratory Interpretations**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

21 **Addicts**
1-2 p.m.—Trenton Psychiatric Hospital
(Trenton Psychiatric Hospital and AMNJ)

22 **Voice of Conscience**
8:30-10:30 p.m.—St. Barnabas Medical Center, Livingston
(New Jersey Psychoanalytic Society and AMNJ)

28 **Ischemic Brain Syndromes**
9-11 a.m.—Riverview Hospital, Red Bank
(Riverview, Dover General, St. Clare's, Denville, and AMNJ)

28 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

March

1 **Improving Psychotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, South Orange
(Group for Advanced Psychiatric Study and AMNJ)

1 **Psychiatric Lecture Series**
8 11 a.m.-12 noon—Greystone Park
15 Psychiatric Hospital
22 *(Greystone Park Psychiatric Hospital and*
29 *AMNJ)*

2 **Psychiatric Lecture Series**
9 1:30-5 p.m.—Trenton Psychiatric
16 Hospital
23 *(Trenton Psychiatric Hospital and*
30 *AMNJ)*

5 **An Adolescent Girl in a Lolie à deux**
8-10 p.m.—111 Ridgewood Ave., Glen Ridge
(Essex Psychiatric Seminar and AMNJ)

5 **Neuroscience Conferences**
12 11:30 a.m.-12:30 p.m.—Bergen Pines
19 County Hospital, Paramus
26 *(Bergen Pines County Hospital and AMNJ)*

6 **Introduction to Family Therapy**
13 7:30-9:30 a.m.—Trenton Psychiatric Hospital
(Trenton Psychiatric Hospital and AMNJ)

6 **Seminar on Law and Psychiatry**
13 3:30-5:30 p.m.—Rutgers Law School,
20 Newark
27 *(Rutgers University Law School and AMNJ)*

7 **Psychological Sequel of Steroid Hormone Exposure**
21 **Mental Health Revolution**
1:30-3 p.m.—NJ Medical School,

Newark
(CMDNJ and AMNJ)

7 **Forensic Psychiatry—Concepts and Areas of Concern**
14 **Forensic Psychiatry—Recent Trends**
21 **Phobic and OBS, Compulsive Neurosis**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

7 **Course for Psychiatrists—Semester II**
14 8-10 p.m.—Hackensack Hospital
21 *(NJ Psychoanalytic Society and AMNJ)*
28

23 **Joint Meeting with New Jersey Psychiatric Association**
8:30-10:30 p.m.—Mayfair Farms, West Orange
(New Jersey Psychoanalytic Society and AMNJ)

23 **Young Freud & Friends—Joint Meeting**
8-10 p.m.—Town & Campus, West Orange
(NJ Psychoanalytic Society, NJ Psychiatric Association and AMNJ)

28 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

28 **Psychopathology of Sexual Dysfunction**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ, and AAFP)

30 **Handling the Patient with Cancer—Psychiatric Considerations**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

OBSTETRICS/GYNECOLOGY

February

1 **Grand Rounds and Case Presentations**
8 4-5 p.m.—Martland Hospital, Newark
15 *(NJ Medical School and AMNJ)*
22

7 **Infertility**
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

7 **Grand Rounds and Case Presentations**
14 2-4 p.m.—Rotating between Martland
21 Hospital, Newark, Newark Beth Israel,
28 St. Michael's, Newark, St. Joseph's Hospital, Paterson and Jersey City Medical Centers
(New Jersey Medical School and AMNJ)

7 **Lectures in Obstetrics and Gynecology**
8-10 p.m.—Location varies
(CMDNJ and AMNJ)

14 **Toxemia of Pregnancy**
11:45 a.m.-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Hospital)

14 **The Laboratory in Gynecology**
8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

20 **Breast Cancer**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

March

1 **Grand Rounds and Case Presentations**
8 4-5 p.m.—Martland Hospital, Newark
15 *(CMDNJ and AMNJ)*
22
29

2 **Annual Edward G. Waters Gynecologic**
4 **Conference**
Begins 8:30 a.m.—Playboy Club, Great Gorge
(CMDNJ and AMNJ)

7 **Lectures in Obstetrics and Gynecology**
8-10 p.m.—Location varies
(CMDNJ and AMNJ)

7 **Grand Rounds and Case Presentations**
14 2-4 p.m.—Rotating between Martland
21 Hospital, Newark Beth Israel,
28 St. Michael's, St. Joseph's Hospital and Jersey City Medical Centers
(CMDNJ and AMNJ)

PATHOLOGY/CLINICAL PATHOLOGY

February

7 **Clinical Pathology Grand Rounds**
14 12 noon-1 p.m.—NJ Medical School,
21 Newark
28 *(NJ Medical School and AMNJ)*

14 **Blood Gases**
1:30-2:30 p.m.—John J. Runnells Hospital, Berkeley Heights
(John E. Runnells Hospital and AMNJ)

27 **Laboratory Interpretations**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

28 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(NJ Medical School and AMNJ)

March

7 **Clinical Pathology Grand Rounds**
14 12 noon-1 p.m.—NJ Medical School,
21 Newark
28 *(CMDNJ and AMNJ)*

PEDIATRICS

February

2 **Advances in Pediatrics**
9 9:30-10:30 a.m.—NJ Medical School,
16 Newark
23 *(NJ Medical School and AMNJ)*

7 **Childhood Leukemia and Solid Tumors**
9-11 a.m.—Riverview Hospital, Red Bank
(Riverview Hospital and AMNJ)

15 **Impact of Genetics on Medical Practice**
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

March

2 **Advances in Pediatrics**
9 9:30-10:30 a.m.—NJ Medical School,
16 Newark
23 *(CMDNJ and AMNJ)*
30

RADIOLOGY

February

8 **Ultrasound Case Presentations**
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

16 **Radiology of the Heart—X-ray, Scan, Echo**
11:45 a.m.-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Hospital)

27 **Ultrasound Diagnosis**
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

March

8 **Case Presentations and Guest Speakers**
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

15 **Combined Radiology and Orthopaedic Meeting**
7:15-10:15 p.m.—Hospital Center at Orange
(Diagnostic Radiology Section for Northern NJ and AMNJ)

SURGERY (General)

February

2 **Head and Neck Center**
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

5 **Lecture Series in Surgery**
12 4:30-5:30 p.m.—NJ Medical School,
19 Newark
26 *(NJ Medical School and AMNJ)*

6 **Weekly Tumor Conferences**
13 11 a.m.-12 noon—Morristown
20 Memorial Hospital
27 *(Morristown Memorial Hospital and AMNJ)*

20 **Breast Cancer**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

21 **Rehabilitation Surgery**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ, and AAFP)

27 **Ultrasound Diagnosis**
8-10 p.m.—The Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

March

5 **Lecture Series in Surgery**
12 4:30-5:30 p.m.—NJ Medical School,
19 Newark
26 *(CMDNJ and AMNJ)*

6 **Weekly Tumor Conferences**
13 11 a.m.-12 noon—Morristown
20 Memorial Hospital
27 *(Morristown Memorial Hospital and AMNJ)*

9 **Carcinoma of the Colon**
14 **CA of the Cervix**
16 **CA of the Urinary Tract**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

20 **Surgical Management—Ulcerative Colitis**
7-8 p.m.—Irvington General Hospital
(Irvington General Hospitl and AMNJ)

27 **Head and Neck Cancer**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

28 **Patient Evaluation for Surgical Procedures**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ, and AAFP)

SURGICAL SPECIALTIES (includes ENT, neurosurgery, ophthalmology, orthopedics, plastics, and vascular surgery)

February

1 **Neurosurgical Case Presentations**
8 4-5:30 p.m.—NJ Medical School,
15 Newark
22 *(NJ Medical School and AMNJ)*

2 **Head and Neck Cancer**
8:30-9:30 a.m.—United Hopitals of Newark
(United Hospitals of Newark and AMNJ)

2 **Diagnostic and Therapeutic Problems in**
9 **Orthopedics**
23 7:30-9 a.m.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital)

March

1 **Neurosurgical Case Presentations**
8 4-5:30 p.m.—NJ Medical School,
15 Newark
22 *(CMDNJ and AMNJ)*
29

6 **Controversial Topics in Esophageal Surgery**
8-10 p.m.—Hilton Hotel, Meadowlands
(N.J. Society of Thoracic Surgeons and AMNJ)

7 **Orthopedic Problems: Bone Tumors**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

10 **Vascular Injuries**
1-5 p.m.—Newark Beth Israel Medical Center
(N.J. Chapter, American Trauma Society, Vascular Society of N.J. and AMNJ)

14 **Current Problems in Transplantation**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ, and AAFP)

14 **Endometrial Carcinoma**
8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

15 **Combined Radiology and Orthopaedic Meeting**
7:15-10:15 p.m.—Hospital Center at Orange
(Diagnostic Radiology Section for Northern NJ and AMNJ)

27 **Vascular Reconstructive Surgery**
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

30 **Fourth Annual Orthopaedic Symposium**
31 8 a.m.—Rutgers Medical School, Piscataway
(CMDNJ and AMNJ)

MISCELLANEOUS

February

7 **Environmental Hazards**
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

27 **Malpractice**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

March

2 **Pharmacology, Clinical**
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

7 **Malpractice**
11:30 a.m.-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

7 **Clinical Pharmacology and Management**
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

7 **Doctors' Time and How to Manage it—**
8:30-11:30 a.m.
Medical Collection Management—
1-4:45 p.m.
Quality Inn, 283 Rt. 17 So., Hasbrouck Heights
(Bergen County Medical Society, AMA and AMNJ)

20 **Orthotics and Prosthetics**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ, and AAFP)

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OBITUARIES

Dr. Preston N. Barton

Preston N. Barton, M.D., a member of our Union County component, died suddenly on September 16, 1978, in Overlook Hospital, Summit. Born in 1913, and graduated from Harvard Medical College, class of 1939, Dr. Barton had practiced in Hartford, Bristol, and Meriden, Connecticut, and was a member of the faculty at Yale University School of Medicine as an instructor in epidemiology and lecturer on occupational medicine. He came to New Jersey in 1967 and opened an office in Mountainside for the practice of occupational and preventive medicine. He was a diplomate of the Board of Preventive Medicine (Occupational Medicine) and a Fellow of the American College o Occupational Medicine.

Dr. Herman J. Horowitz

A member of our Bergen County component, Herman J. Horowitz, M.D., died on September 21, 1978, in Hackensack Hospital of an apparent heart ailment. A graduate of the University of Maryland's School of Medicine in 1922, Dr. Horowitz had practiced surgery in Bergen County for many years, having been affiliated with Englewood Hospital and Holy Name Hospital in Teaneck. He was a Fellow of the American College of Surgeons and a member of the Englewood Surgical Society. During World War II Dr. Horowitz served for four years with the medical department of the United States Army. He was 79 years old at the time of his death.

Dr. Morris Joseph

On October 6, 1978, Morris Joseph, M.D., an emeritus member of our Passaic County component, formerly of Passaic, died in Wichita, Kansas after a long illness. A graduate of Johns Hopkins Medical School, class of 1915, Dr. Joseph had practiced general and gynecologic surgery and had been on the staff at Passaic General Hospital. He was a Fellow of the American College of Surgeons. Dr. Joseph was 89 years old at the time of his death.

Dr. Sigmund S. Keil

Sigmund S. Keil, M.D., a senior member of our Union County component, died on October 27, 1978. A native of New York City, born in 1908, Dr. Keil was graduated from the University of Vermont Medical School, class of 1935, and pursued graduate work in cardiology and pediatrics at New York Polyclinic Hospital. He practiced general medicine with special interest in pediatrics in Linden for many years. He had been affiliated with St. Elizabeth Hospital in Elizabeth. Dr. Keil was active in civic affairs and was chief medical officer for the Linden schools.

Dr. Jacob H. Lefkowitz

Word has just been received of the death, from a heart ailment, of Jacob H. Lefkowitz, M.D., on September 7, 1978, in Englewood Hospital. Born in Russia in 1899, Dr. Lefkowitz earned his medical degree from Long Island University College of Medicine in 1926 and pursued graduate work in pediatrics, becoming board certified in that specialty. He had practiced in West New York and was on the staff at Christ Hospital in Jersey City.

Dr. Thomas J. Ormsby

Thomas J. Ormsby, M.D., formerly of Newark, and a member of our Essex County component, died on October 24, 1978, in Pompano Beach, Florida, where he was living in retirement. Born in 1906 and graduated from New York University Medical College, class of 1934, Dr. Ormsby practiced internal medicine, with special interest in chest diseases, in Essex County until his retirement in 1970. He had been affiliated with St. Michael's Hospital in Newark, the New Jersey Orthopedic Hospital in Orange, and Clara Maass Memorial Hospital in Belleville. He was a Fellow of the American College of Chest Physicians and a member of the Academy of Medicine of New Jersey.

Dr. Arthur B. Peacock

A senior member of the Burlington County component, Arthur B. Peacock, M.D., died on October 29, 1978. A native of Rhode Island, Dr. Peacock earned his medical degree from Jefferson Medical College in 1931 and took graduate work in internal medicine before opening an office for the practice of that specialty, first in Columbus and later in Moorestown where he remained until retirement in 1975. Dr. Peacock had been president of his county medical society and a member of the New Jersey State Board of Health and of the Moorestown Board of Health. He had been affiliated with Burlington County Memorial Hospital in Mount Holly.

Dr. Juan B. Pulido

Word has just been received of the sudden and untimely death of Juan B. Pulido, M.D., in Englewood Hospital on August 8, 1978. Born in Cuba in 1929, Dr. Pulido was graduated from the University of Havana School of Medicine in 1954 and pursued graduate work in pediatrics and orthopedics at the same institution. He emigrated first to Puerto Rico in 1961 and later the same year to New Jersey. After serving as resident physician in several New Jersey hospitals, he established a pediatric practice in Newark and became a member of our Essex County component. He was on the attending staff at St. Michael's and St. James' Hospitals in Newark and was assistant medical director of the children's unit at the New Jersey State Hospital at Greystone Park.

Dr. Harry Schreck

On September 19, 1978, at St. Barnabas Medical Center in Livingston, Harry Schreck, M.D., a member of our Essex County component, died suddenly after a coronary attack. A native of Austria, born in 1903, Dr. Schreck earned his medical degree from New York University, class of 1927, and had practiced general surgery in Newark for many years. He had been affiliated with Martland and Presbyterian Hospitals there and was a Fellow of the American College of Surgeons.

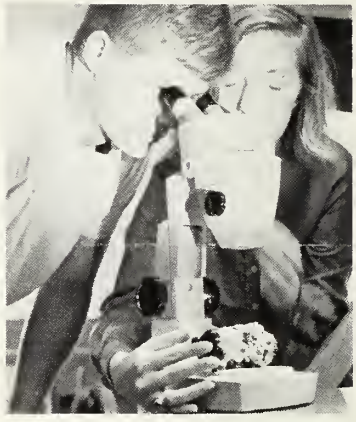
Dr. Hyman J. Udinsky

One of Passaic County's emeritus members, Hyman J. Udinsky, M.D., died on October 4, 1978, in Miami Beach, Florida, where he was living in retirement. Born in 1888, Dr. Udinsky was graduated from Jefferson Medical College in 1912 and pursued graduate work in dermatology, which specialty he practiced in Passaic for many years, retiring in the early 60's.

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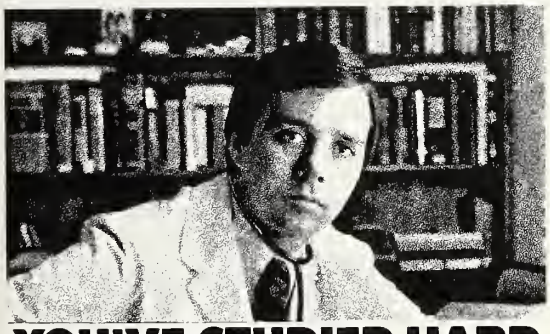
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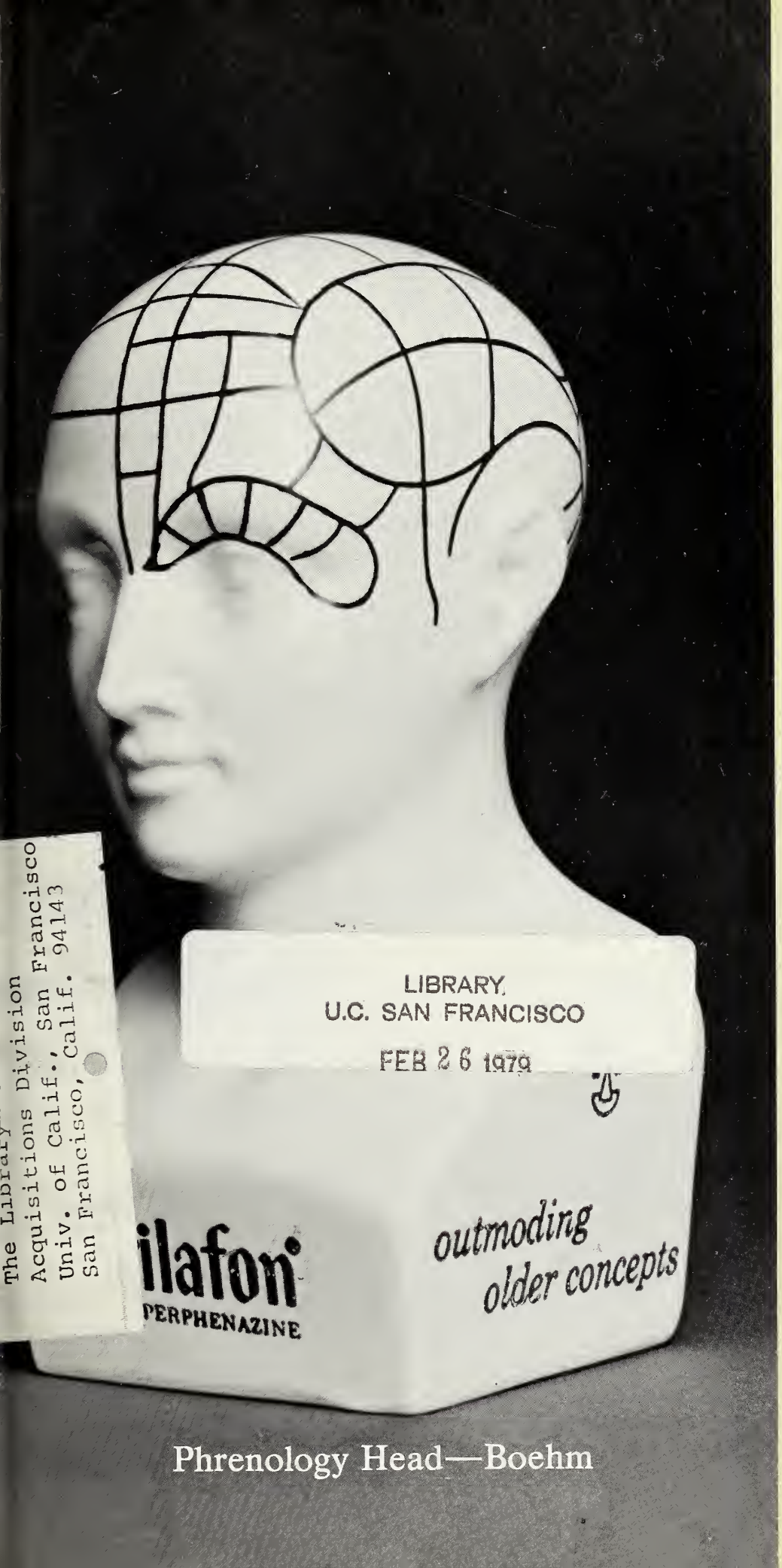
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E. J. Lazaro, M.D., et al.

Mystery and Magic in Guise of Déjà Vu
Steven R. Kohn, M.D.

Surgical Treatment of Glaucoma
A. A. Cinotti, M.D.
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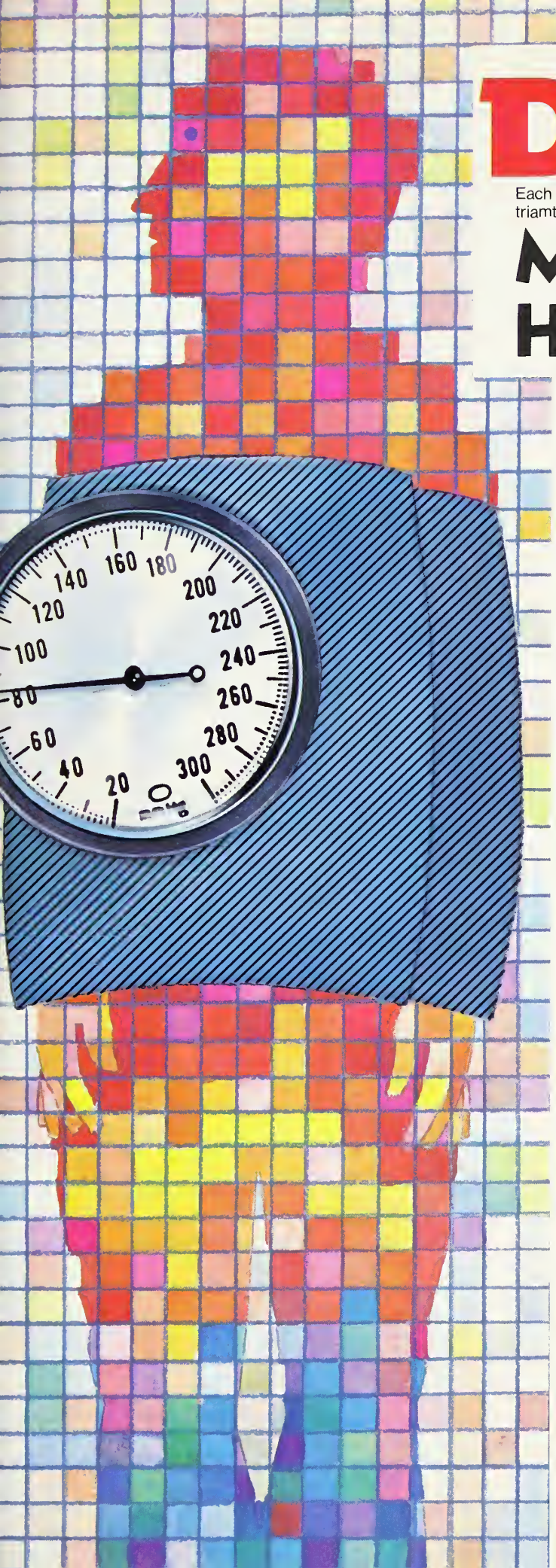
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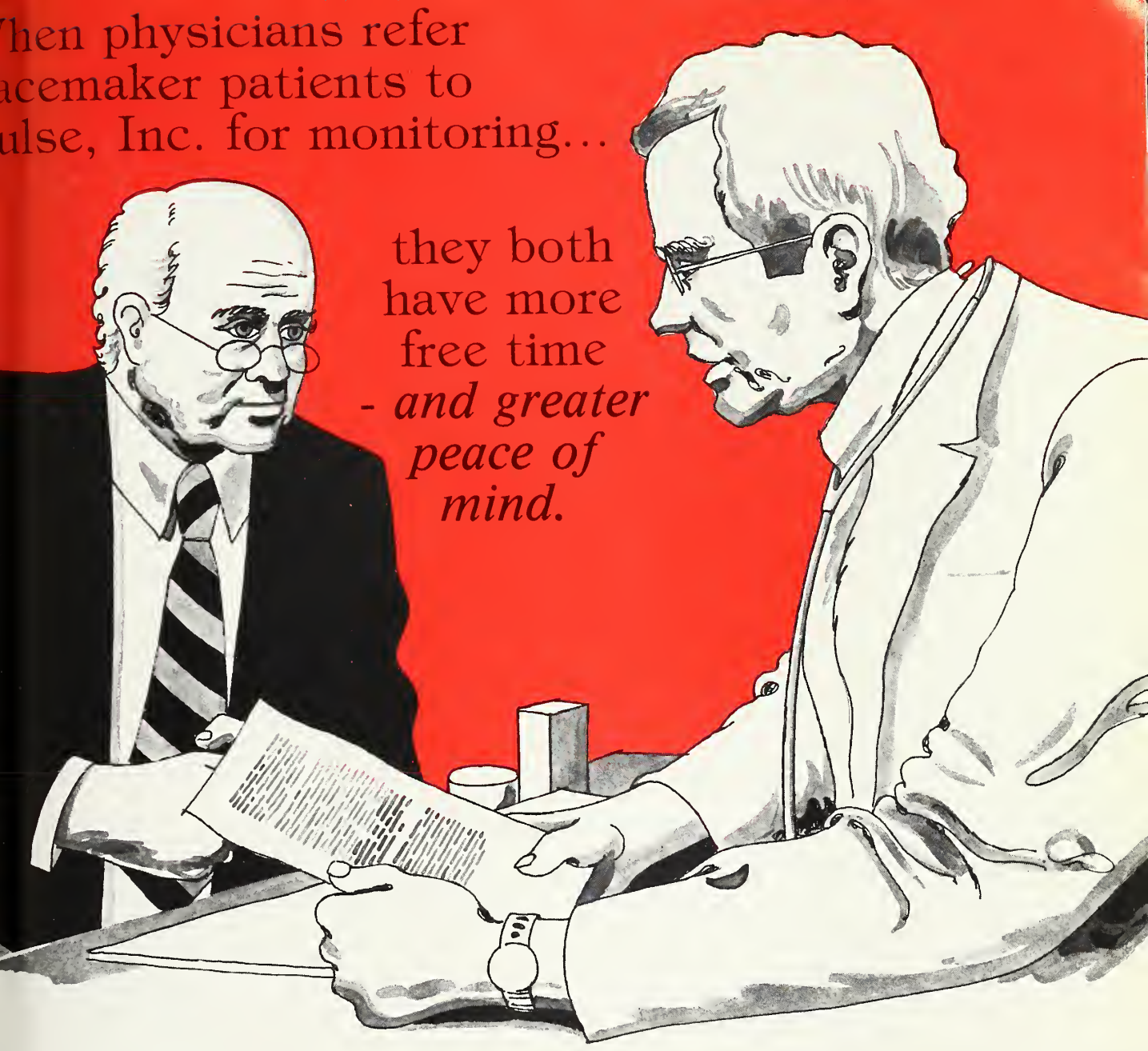
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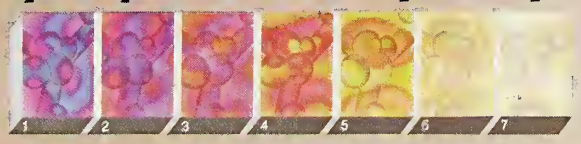
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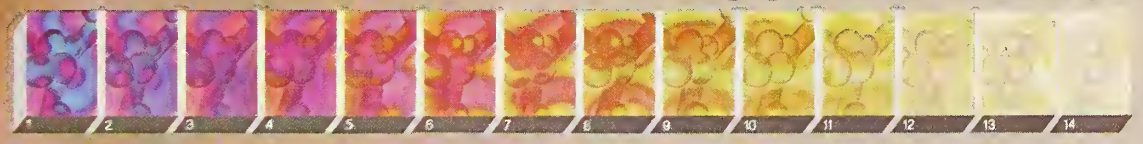
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All of us must work together to help contain healthcare costs.



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Accident Facts. Chicago, Illinois, National Safety Council, 1974.

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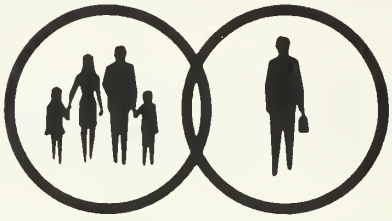
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The Phrenology Head

The cover of this issue shows a valuable collector's piece, which some of the readers of this *Journal* may own and not recognize as the work of the late Edward Marshall Boehm. The internationally renowned porcelain artist opened his first studio at 600 Stokes Avenue in Trenton in 1950 and went on to help reestablish Trenton as one of the great porcelain centers of the world. The total output of this remarkable sculptor prior to his premature death in 1969 is legendary; his creations have been owned by presidents, kings, and royalty of many nations, by Popes John and Paul VI, as well as by the most important museums in the world.

The Phrenology Head was a product of Boehm's early days when he accepted private commissions and requests from "firms in search of fine promotional pieces." There were four versions of the Phrenology Head as described in the following catalog quotation[1]:

The RPC-700-03 (Trilafon) and 04 (Etrafon) were commissioned by the Schering Corporation in quantities of approximately 5,000 each. They are 5-1/4" high and the later model is sectionalized by incised lines. Both bear makersmark D.

"The sculpture (03) is a bust with head divided into sections by black lines inscribed on the front 'Trilafon Perphenzine,' lines inscribed on the right side 'Schering, full-range tranquilizer,' and on the left side 'Schering, outmoding older concepts.'

"There was indeed another Phrenology Head done, but not by Boehm. This has led to a good deal of confusion and there is only one rather subtle way of detecting the difference between them. When Schering first commissioned the RPC-700-03, they ordered 5,000 pieces. The promotion for Schering was an extremely successful one and there was an immediate demand for additional pieces by physicians. Schering went back to Boehm, and at that time Boehm was not prepared to do any more and Schering turned to another Trenton house which turned out an additional 5,000 on Boehm's original molds. This is the RPC-700-06. It is difficult to tell them apart, but if they are turned upside down, the original Boehm Phrenology Head (that is, the Trilafon Head) has a slight ridge along the bottom around the base with an indentation at the center. The Trilafon Head by the other potter (RPC-700-06) has a completely flat base. The head done by the other potter has only a curiosity value, whereas the Trilafon is a genuine collector's object.

"The RPC-700-05 Phrenology Head, which was labeled as the Bald Man, carries an interesting story. It was done by Ed Boehm as a special gift for a friend of his. The friend was, as you may have guessed, bald."

Phrenology was the name given to a pseudo-science formulated by Franz Joseph Goll about 1800. The concept was based on the belief that there was strict localization of function in the human brain. Goll extended the notion that each mental faculty had its focal point in a definite region of the surface of the cerebrum to the extrapolation that there was created a correspondent anatomical change in the skull. Thus, the phrenologist could evaluate the personality and the abilities of his subject by digital examination of the head and, in particular, the "bumps" on the skull.

Phrenology enjoyed popular appeal for a time, but was later discredited by scientific research, which resulted in disgrace and vituperation for Goll and his colleague Johann Caspar Spurzheim.

The idea of brain function localization traces back very far, but serious interest in it began two centuries ago. According to Wechsler, Goll "was a good physiologist and even better anatomist. . .who specifically localized intelligence and the emotions in the brain."[2]

It was remarkable that researchers at the beginning of the last century worked on the theory of localization "by exposing the brain, testing isolated parts, and establishing the excitablity of the cortex."[2]

It is also remarkable that research workers are doing the same thing today. A neuropsychologist-neurolinguist, Harry Whitaker, at the University of Rochester, has been mapping the sites of language in the brain. By working with the exposed brain during neurosurgical procedures, Whitaker and his associates pinpoint the cortical locations of language by subjecting patients to visual or verbal tasks while interfering with brain function by delivering a weak electrical current to two points on the brain by a pair of silver electrodes separated by a distance of five millimeters. Whitaker has found that an unexpectedly large amount of the left hemisphere is devoted to language and that different languages occupy different cerebral localizations in multilingual individuals.

Progress is not always expeditious and time has a way of clouding some issues. Goll was confused in his notion of phrenology, but he was dead right in his theories of functional localization in the brain.

Look around among your possessions for the Phrenology Head. If you have one, think kind thoughts about Edward Marshall Boehm, a great artist, and Franz Joseph Goll, "a good physiologist and even better anatomist." A.K.

References

[1] Palley R: *The Porcelain Art of Edward Marshall Boehm.* New York, Harry N. Abrams, 1976.

[2] Wechsler IS: *A Textbook of Clinical Neurology.* Philadelphia, Saunders, 1947, pp. 763-764.

The House Has Spoken*

On Sunday, December 19, 1978, the House of Delegates of the Medical Society of New Jersey, at a special session, voted to continue to oppose the licensure and/or registration of physicians' assistants.

This matter has been reviewed and rereviewed by official bodies of the Medical Society of New Jersey over the past several years. No stones have been left unturned to gain a clear picture of the issues. The House of Delegates, in May, 1978, adopted Resolution #9 which stated:

"Resolved that the Medical Society of New Jersey continue to oppose the licensure and/or registration of physicians' assistants because no definite need has been established."

Because of the progress made in the development of proposed regulatory acts (Assembly Bill 1387 and Senate Bill 1192) by the New Jersey State Assembly and Senate, MSNJ President Krueger appointed an Ad Hoc Committee on Physicians' Assistants to reexamine the issues very carefully. The committee met twice, conducted a telephone poll of one percent of the membership and then passed the following motion by a vote of three to two:

To oppose the premise of physicians' assistant legislation because of:
(1) the present position (May 1978) of the House of Delegates against the concept of physicians' assistants, and
(2) the failure of a proven need for physicians' assistants.

The *majority report* summarized its reasons as follows:

1. No one has proved the need for creating a second-class helper to do a first-class job; because, in spite of all arguments in his favor, the physicians' assistant *will* be practicing medicine without the full training of an M.D. or D.O.
2. No one has proved that the population as a whole, or any group of patients in particular, would rather be examined, injected, immunized, sutured, counseled, educated, and referred by physicians' assistants than by a licensed physician.
3. And, in spite of the fact that none of the physicians' assistant legislation mandates their use by physicians or hospitals, no one has proved that permission to do so will upgrade the quality of medical care in New Jersey.

The author of the *minority report* of the Ad Hoc Committee, despite several serious defects in proposed bills A-1378 and S-1192, recommended "conditional approval." His reasons for supporting the physicians' assistant concept are:

1. There are many physicians in this state who would utilize the services of a physicians' assistant. It is of little importance as to whether or not their numbers are in the majority. It is important, however, that their numbers are considerable. The proposed legislation is an enabling legislation and there is nothing in it that states that any of us must use them.
2. Professionally trained physicians' assistants are widely accepted throughout the United States. They are approved by all the major professional organizations. Federal and state governments fund their training programs.
3. Physicians' assistants should be thought of as physician extenders. They may assist the primary care physician, or they may assist the specialist. Most of us feel that their use as surgical assistants would be welcomed.
4. In this state, physicians' assistants have the backing of most health-related groups and probably the vast majority of legislators.
5. Many physicians have described their experiences with physicians' assistants. By and large these experiences have been favorable. Most of the younger physicians have worked with them in medical schools and residency programs, and have come to *expect* their assistance.
6. Many of us fear independent physicians' assistant practice. There is no state in the union which allows this and, up to this point, there is no indication that it will be a problem.
7. It is well demonstrated that their (physicians' assistants) services can make the physicians' services more efficient and, therefore, do allow the physician to serve his patients better.

At a regular meeting on October 15, 1978, the Board of Trustees reversed the recommendations of the Council on Legislation and the Ad Hoc Committee on Physicians' Assistants and voted to adopt (12 to 5) the position on A-1192 and A-1387 of *conditional approval* "requiring modifications to preserve quality care and reduced costs." The Board also voted to defeat (7 to 10) a motion calling for a special session of the House of Delegates to discuss current legislation on physicians' assistants.

Despite this action of the Board, a grass-roots petition at the county level required the President of MSNJ to call a special session of the House. He did so.

At this session, which was "highly emotional," the House overwhelmingly voted to maintain a position of *active opposition* to the proposed regulations dealing with physicians' assistants. It appeared that those delegates who opposed the PA legislation had strong genuine concerns, while those who favored the concept and the proposed bills were less demonstrative vocally.

In the final analysis, the House spoke with a strong and unified voice, which originated at the grass-roots level, and reversed the position of its leadership. Responding to this democratic decision, both President Krueger and the Board of Trustees have developed a strategy to be implemented to bring the feelings of the Medical Society of New Jersey and its membership to the collective and individual legislators in the New Jersey Assembly and Senate. This will be done.

Whether the proposed bills become law or not, this Medical Society has investigated and debated the issues and reached its conclusions in a democratic fashion. Whether the physician members of this Society agree with the decision of its delegates and leaders, they can be proud of their forthrightness, seriousness of purpose, and, in the final analysis, their primary interest in the care of the patient. A.K.

*All of the points brought out in this editorial have been covered in *The Journal* in previous issues: 75:Tr61 (July Transactions) 1978; 75:942-943 (December) 1978; 76:58-59 (January) 1979.

Phencyclidine (Angel Dust, PCP) is an easily synthesized, widely abused, and dangerous drug. Its ingredients are obtained readily from chemical supply firms, and the compound can be synthesized by amateur chemists in apartment kitchens or bathrooms. Its ready availability made phencyclidine the usual street substitute for LSD, THC, mescaline or psilocybin during the 1960s, and more recently it has achieved fame under its own name. Frequent users claim that it provides a more pleasant "trip" than that of marihuana, and although most users never require medical attention, toxic reactions can occur and may closely mimic schizophrenia.

Like ketamine, to which it is structurally related, phencyclidine is a dissociative anesthetic which reduces the effect of sensory input to the cortex, and produces hallucinations, disorientation, and confusion. Low doses produce disinhibition with euphoria and a feeling of walking or floating on clouds; behavior becomes looser and the emotions more labile. Some users report LSD-like effects such as slowing of time, feelings of power, illusions and pseudo-hallucinations. Overdose can lead to stupor or coma. This may be followed by delirium with paranoia, auditory hallucinations, disorientation, restlessness, and violent assaultiveness, or the delirium may appear without preceding stupor or coma.

A schizophreniform psychosis may be seen after one or several exposures to PCP. This is more likely to occur in persons who had previous schizophrenic episodes but many users have developed severe and long-lasting psychoses without such a history. Acute paranoid or catatonic schizophrenia may be closely simulated and it is advisable that urines of patients with such clinical pictures be tested for PCP and amphetamines. After the psychotic state recedes (and this may take many weeks) depression with suicidal impulses may appear.

Concern about the abuse of PCP led the National Institute on Drug Abuse to hold a conference of major investigators in Monterey, California last February. One outcome of this conference is the review in Doctors' Notebook this month. (see pages 139-144) which summarizes what is known about phencyclidine and its effects. The authors have disappointingly little to say about methods of preventing or controlling the spreading abuse of this dangerous drug. Their review provides a clear and concise account of the clinical pictures seen in phencyclidine users and describes how they best may be treated. It is recommended as essential reading for psychiatrists, emergency room physicians, and parents.

A. Arthur Sugerman, M.D.

213th Annual Meeting—Atlantic City

PRESIDENT'S INAUGURAL RECEPTION

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The evidence of experience

Since October 1974 when Motrin® (ibuprofen) was introduced in the United States, it has been used by more than 6,000,000 patients with rheumatoid arthritis* or osteoarthritis. Rarely has an ethical pharmaceutical product been prescribed for so many patients in so short a time. In addition, more than 450 studies presenting new data related to Motrin have been published.

The 6,000,000 patients already treated with Motrin is an objective measure of physicians' confidence in the ability of Motrin to relieve the pain and inflammation associated with rheumatoid arthritis and osteoarthritis.

So it is not surprising that in this short period Motrin has become the most frequently prescribed alternative to aspirin. Motrin relieves joint pain and inflammation as effectively as indomethacin or aspirin, but causes significantly fewer CNS and milder GI reactions. However, gastrointestinal bleeding, sometimes severe, has been associated with Motrin, aspirin, indomethacin, and other nonsteroidal antiarthritic agents.

*The safety and effectiveness of Motrin have not been established in patients with Functional Class IV rheumatoid arthritis (incapacitated, largely or wholly bedridden, or confined to wheelchair; little or no self-care).

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The confidence that comes from experience—one more reason to prescribe Motrin.

Please turn page for a brief summary of prescribing information.

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Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin (ibuprofen) is gastrointestinal (4% to 16%). This includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness*, headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

Incidence: Unmarked 1% to 3%; *3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

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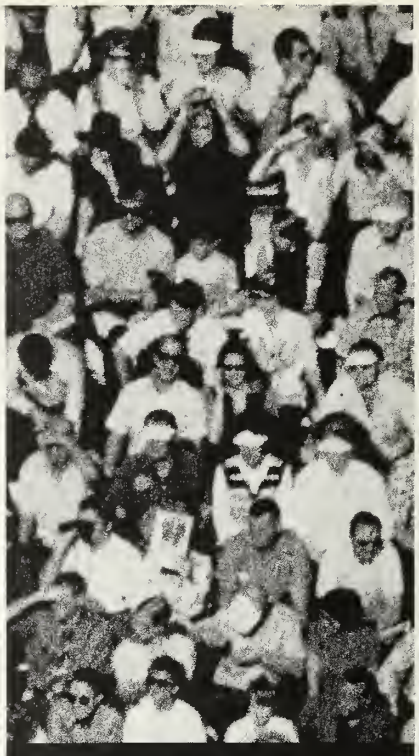
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Controversies in the Management of Cancer of the Colon and Rectum: A Questionnaire Course

E.J. LAZARO, M.D., B.F. RUSH, JR., M.D.,
A.P. SWAMINATHAN, M.D., E.L. GRIMES, M.D.
AND D.K. BRIEF, M.D., Newark.

The responses to a questionnaire on "Controversies in the Management of Cancer of the Colon and Rectum" are reported and discussed.*

In this communication we present the responses to the Questionnaire Course on "Controversies in the Management of Cancer of the Colon and Rectum". This course constituted part of a seminar on colorectal cancer that was presented at the 212th Annual Meeting of the Medical Society of New Jersey held at Atlantic City on May 8, 1978. Details of this course that was jointly sponsored by the New Jersey Chapter of the American College of Surgeons, the Medical Society of New Jersey, the Academy of Medicine of New Jersey and the College of Medicine and Dentistry of New Jersey have been outlined in previous reports.[15]

The preferences to the various alternatives indicated for each item in the questionnaire are contained in the following tabulation.

Questions	*Preferred Answers of Respondents (%)*
1. You employ the "no-touch, isolation technique" for resection of carcinoma of the colon	
(1) never	11
(2) always or almost always	35
(3) sometimes	43
(4) frequently now, although you did not do so earlier in your career	11
(5) no opinion	0
2. The number of patients with carcinoma of the rectum that you have treated by electrocoagulation (fulguration) is	
(1) none	36
(2) less than five	50
(3) between five and twenty	11
(4) over twenty	2
(5) no opinion	1
3. In your practice, use of postoperative chemotherapeutic drugs following colectomy for cancer is	
(1) routine for all or most cases	7
(2) restricted to patients with positive nodes	55
(3) routine for all patients except those with Dukes' A lesions	16
(4) seldom or never indicated	22
(5) no opinion	0
4. Ligation of the inferior mesenteric vessels at their origin has been recommended as an early step in anterior resection of cancer of the distal colon and rectum. You	
(1) have previously employed this technical step but do not do so any more	22
(2) have always employed this technical step	34
(3) have never resorted to this technical step	17

*Presented before the joint session of the Sections on Clinical Pathology, Gastroenterology and Proctology and Surgery at the 212th Annual Meeting, the Medical Society of New Jersey, May 8, 1978, Atlantic City. Dr. Lazaro is Professor of Surgery; Dr. Rush is Chairman and Johnson and Johnson Professor of Surgery; Dr. Swaminathan is Assistant Professor of Surgery; Dr. Brief is Clinical Associate Professor of Surgery, at New Jersey Medical School, CMDNJ, Newark. Dr. Grimes is Chief of Surgery, Our Lady of Lourdes Hospital, Camden. Dr. Lazaro may be addressed at Martland Hospital, CMDNJ, 65 Bergen Street, Newark, NJ 07107.

Questions	Preferred Answers of Respondents (%)
(4) frequently or always use this technical step, although you did not do so previously	24
(5) have no opinion	3
5. You employ *preoperative* irradiation for carcinoma of the colon and rectum	
(1) never, because it may jeopardize healing of the anastomosis	21
(2) sometimes, in order to shrink the tumor	21
(3) frequently, because it improves the survival rate	1
(4) only for selected cases of *rectal* lesions	53
(5) no opinion	4
6. In your work-up of a patient with carcinoma of the colon, you obtain liver scans to detect hepatic metastases	
(1) never	5
(2) occasionally	30
(3) frequently	57
(4) infrequently or never now, although you often did so earlier in your career	8
(5) no opinion	0
7. A patient has a 4 cm broad-based villous adenoma in the distal part of the extraperitoneal rectum. Multiple biopsy specimens show only superficial or *in situ* carcinoma (there is no invasion of the muscularis mucosae). You are most likely to manage this patient by	
(1) abdominoperineal resection	15
(2) electrocoagulation	27
(3) transanal resection	34
(4) resection through the Kraske approach (posterior trans-sacral)	18
(5) another method, you have no opinion	6
8. Following abdominoperineal resection for carcinoma of the rectum, you prefer to manage the perineal wound by	
(1) primary closure without drainage	2
(2) primary closure with Penrose drains	34
(3) primary closure with sump drains in the perineal space	46
(4) gauze packing of the perineal space with or without secondary closure later	17
(5) another method or you have no opinion	1
9. Your attitude towards colonoscopy is best summarized by which one of the following statements?	
(1) You perform colonoscopy on your patients yourself	12
(2) You generally refer your patients requiring colonoscopy to another surgeon	12
(3) You refer your patients requiring colonoscopy to a gastrointestinal (medical) endoscopist	70
(4) You never, or almost never, request colonoscopy on your patients	6
(5) Another statement, or you have no opinion	0
10. A 10-year-old girl is diagnosed as having familial polyposis of the colon. You preferably would manage this patient with	
(1) total colectomy, abdominoperineal resection of the rectum and ileostomy	7
(2) total colectomy, ileoproctostomy, and frequent transanal endoscopies for the rest of her life	57
(3) total colectomy and ileoproctostomy now, and abdominoperineal resection with ileostomy a few years later	8
(4) no surgical treatment till some years later	23
(5) another plan, or you have no opinion	5
11. In the preparation of a patient with carcinoma of the colon for surgery, the antibiotic agent you are least likely to employ is	
(1) erythromycin	17
(2) neomycin	8
(3) a tetracycline	42
(4) cephalothin (Keflin)®	27
(5) another agent or you have no opinion	6

Questions	Preferred Answers of Respondents (%)
12. A 45-year-old female has a sessile carcinoma, 3 cm in diameter and 15 cm from the anal verge on the anterior colonic wall. There are no detectable metastases elsewhere. Which of the following procedures are you most likely to perform on this patient?	
(1) Anterior resection with total abdominal hysterectomy	23
(2) Anterior resection only	75
(3) Abdominoperineal resection only	1
(4) Abdominoperineal resection with hysterctomy	1
(5) No opinion	0
13. Pull-through procedures have been recommended by some authors following resection of a malignant rectal lesion in order to avoid a colostomy. With respect to this technique you	
(1) have no experience	74
(2) employed it at one time but then abandoned it	15
(3) use it occasionally	9
(4) use it frequently	0
(5) have no opinion	2
14. Following hemorrhoidectomy in a 60-year-old female, the pathologist reports carcinoma *in situ* in one of the specimens. You feel that this patient should now	
(1) undergo abdominoperineal resection	8
(2) be placed on cancer chemotherapy	0
(3) be followed by periodic examinations	65
(4) undergo wider removal of the hemorrhoidal areas	26
(5) no opinion	1
15. A 55-year-old male undergoes colonoscopic removal of a pedunculated polyp 3 cm in diameter in the sigmoid colon. On histological examination the lesion is found to be malignant, and there is invasion of the stalk. You would advise this patient initially to have	
(1) cancer chemotherapy	0
(2) irradiation	0
(3) segmental colectomy	96
(4) colonoscopic examination at periodic intervals	4
(5) no opinion	0
16. A 50-year-old male, who is a good surgical risk, is found on barium enema examination to have a pedunculated polyp, 2 cm in diameter, in the mid-transverse colon. Preferably you would advise him to undergo	
(1) segmental colectomy	7
(2) colostomy with polypectomy	18
(3) colonoscopic removal of the polyp	74
(4) continued observation with periodic colonoscopic or barium enema examinations	1
(5) another option, or you have no opinion	0
17. A 55-year-old woman has a large soft tumor occupying two-thirds of the circumference of her distal rectum and extending to 3 cm from the dentate line. The anterior segment of the rectum appears free of tumor. Multiple biopsies show villous adenoma but one section was suggestive of carcinoma *in situ*. You are most likely to initially recommend this patient to have	
(1) abdominoperineal resection	39
(2) pull-through resection of the rectum	5
(3) snare excision of the lesion with electrocoagulation	31
(4) resection of the lesion through a posterior approach	21
(5) another option or no opinion	4
18. Some groups recommend synchronous abdominoperineal resection utilizing a two-team approach, one team for the abdominal, and the other team for the perineal aspects of the procedure. You	
(1) customarily use this approach	1
(2) have used this approach at one time but subsequently abandoned it	23
(3) were initially opposed to this approach but now find it helpful	4
(4) have never used this approach	52
(5) have no opinion	2

Questions	Preferred Answers of Respondents (%)
19. As a temporary decompressive procedure you employ cecostomy	
(1) never	30
(2) sometimes	54
(3) frequently	15
(4) always	1
(5) no opinion	0
20. You employ staplers for colonic anastomosis	
(1) always, or almost always	9
(2) frequently	12
(3) sometimes	22
(4) seldom or never	55
(5) no opinion	2
21. Following anterior resection of the distal colon for carcinoma you employ a temporary transverse colostomy	
(1) always	2
(2) frequently	9
(3) sometimes	35
(4) seldom or never	54
(5) no opinion	0
22. Your procedure of choice in the management of a poor-risk patient with an obstructing carcinoma of the left colon is	
(1) initial transverse colostomy followed 10-14 days later by resection and when suitable, colostomy closure, (3 stage)	57
(2) primary resection with colostomy and distal mucous fistula followed by closure of the colostomy (2 stage)	29
(3) primary resection and anastomosis (1 stage)	8
(4) other approach	5
(5) no opinion	1
23. Your procedure of choice for a patient with carcinoma of the right colon with 2 polyps in the transverse colon, 3 scattered polyps in the descending colon, 2 polyps in the proximal sigmoid colon and a normal rectum is:	
(1) right hemicolectomy with colonoscopic removal of the other polyps	22
(2) right hemicolectomy with intraoperative removal of the other polyps by multiple colostomies	20
(3) subtotal colectomy and ileoproctostomy	57
(4) abdominoperineal total proctocolectomy and ileostomy	1
(5) other procedure or no opinion	0

COMMENT

Surgical practice is to a great extent dictated by professional attitudes that are in vogue at a particular time. For this reason, analysis of the responses to this questionnaire can, by indicating the more common approaches to various aspects of colorectal cancer, provide appropriate guidelines for the current management of this disease.

There are several areas in which most of our respondents share a common opinion concerning a variety of diagnostic and therapeutic items. Electrofulguration of carcinoma of the rectum is not a popular modality of therapy. Following colectomy for cancer of the large bowel, cancer chemotherapy is more likely to be employed in patients with positive nodes. In abdominoperineal resection for carcinoma of the rectum, the perineal wound usually is closed primarily together with the employment of Penrose or sump drains. Most surgeons refer their patients who require colonoscopy to a medical endoscopist (gastroenterologist). Colonscopic polypectomy is the preferred initial procedure for a patient with a two cm pedunculated colonic polyp. In a female with a sessile carcinoma on the anterior colonic wall and 15 cm from the anal verge without detectable metastases, most surgeons would opt for anterior resection without including a hysterectomy in the procedure. Pull-through operations for the resection of malignant rectal lesions in order to avoid a colostomy are not utilized by the majority of our respondents. Synchronous abdominoperineal resection utilizing a two-team approach is favored by only 23 percent of our surgeons. Either cecostomy or colostomy for temporary post-colectomy decompression are seldom considered necessary in elective colectomy. Staplers are infrequently employed for carrying out colonic anastomosis. And, a poor-risk patient with obstructing carcinoma of the left colon is more likely to be managed in three surgical stages.

As we have indicated in our early experience with the questionnaire course, there are certain situations in which two or sometimes three different approaches are considered to be equally acceptable in surgical therapeutics.[1] These situations which are revealed in our questionnaire include: (1) use of the "no-touch isolation" technique for the resection of colonic cancer; (2) high ligation of the mesenteric vessels as an initial step in anterior resection; (3) employment of preoperative irradiation for cancer of the colon and rectum; and (4) the choice of procedure(s) for a patient with carcinoma of the right colon together with a few polyps scattered through the colon and rectum.

The main purpose of the questionnaire course is to assess the current status of practice pertaining to a selected surgical topic.[1-3] That attitudes in the management of a disease are likely to change within a period of a few years was pointed out in two surveys on cancer of the breast, one held in 1971 and the other in 1977.[5] It is, therefore, conceivable that significant changes will take place in the management of certain aspects of cancer of the colon and rectum, and will be reflected in a repeat survey held some years later. Finally, it should be pointed out that, since there is a lack of data similar to ours on a regional or national basis, any comment concerning how surgical practice compares with that in other geographic areas is not possible.

SUMMARY

The responses to a questionnaire on "Controversies in the Management of Cancer of the Colon and Rectum" which constituted part of a seminar held in Atlantic City, New Jersey on May 8, 1978 at the 212th Annual Meeting of the Medical Society of New Jersey are reported. Areas in which most surgeons shared the same opinion and those in which two, or sometimes three, opinions were recorded with approximately equal frequency are noted.

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DESCRIPTION: Methyltestosterone is 17β-Hydroxy-17-Methylandrost-4-en-3-one. **ACTIONS:** Methyltestosterone is an oil soluble androgenic hormone. **INDICATIONS:** In the male: 1. Eunuchoidism and eunichism 2. Male climacteric symptoms when these are secondary to androgen deficiency. 3. Impotence due to androgenic deficiency 4. Post-puberal cryptochidism with evidence of hypogonadism. Cholestatic hepatitis with jaundice and altered liver function tests, such as increased BSP retention, and rises in SGOT levels, have been reported after Methyltestosterone. These changes appear to be related to dosage of the drug. Therefore, in the presence of any changes in liver function tests, drug should be discontinued. **PRECAUTIONS:** Prolonged dosage of androgen may result in sodium and fluid retention. This may present a problem, especially in patients with compromised cardiac reserve or renal disease. In treating males for symptoms of climacteric, avoid stimulation to the point of increasing the nervous, mental, and physical activities beyond the patient's cardiovascular capacity. **CONTRAINDICATIONS:** Contraindicated in persons with known or suspected carcinoma of the prostate and in carcinoma of the male breast. Contraindicated in the presence of severe liver damage. **WARNINGS:** If priapism or other signs of excessive sexual stimulation develop, discontinue therapy. In the male, prolonged administration or excessive dosage may cause inhibition of testicular function, with resultant oligospermia and decrease in ejaculatory volume. Use cautiously in young boys to avoid premature epiphyseal closure or precocious sexual development. Hypersensitivity and gynecomastia may occur rarely. PBI may be decreased in patients taking androgens. Hypercalcemia may occur, particularly during therapy for metastatic breast carcinoma. If this occurs, the drug should be discontinued. **ADVERSE REACTIONS:** Cholestatic jaundice • Oligospermia and decreased ejaculatory volume • Hypercalcemia particularly in patients with metastatic breast carcinoma. This usually indicates progression of bone metastases • Sodium and water retention • Priapism • Virilization in female patients • Hypersensitivity and gynecomastia. **DOSAGE AND ADMINISTRATION:** Dosage must be strictly individualized, as patients vary widely in requirements. Daily requirements are best administered in divided doses. The following is suggested as an average daily dosage guide. **In the male:** Eunuchoidism and eunuchism, 10 to 40 mg.; Male climacteric symptoms and impotence due to androgen deficiency, 10 to 40 mg.; Postpuberal cryptorchism, 30 mg. **REFERENCE:** R. B. Greenblatt, M.D.; R. Witherington, M.D.; I. B. Sipahioglu, M.D.: Hormones for Improved Sexuality in the Male and the Female Climacteric. *Drug Therapy,* Sept. 1976. **SUPPLIED:** 5, 10, 25 mg. in bottles of 60, 250. Rx only.

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The Hematofluorometer in Screening for Lead Poisoning

STEVEN M. MARCUS, M.D. and
ROBERT ZIERING, M.D., Newark*

Comparisons were made of various methods for lead intoxication screening-testing. The hematofluorometer, placed into field use, produced reliable, consistent results.

As mass screening for children with lead poisoning increases, the need for quick and reliable identification of those children who require intervention becomes apparent. The micro method, Free Erythrocyte Protoporphyrins (FEP), [1-4] has become the main thrust in this direction. In this institution's experience, as our lead program developed and as other institutions joined in submitting samples to the central state lead laboratory, the turnaround time from sampling to results has lengthened from two to fourteen days. The population attending inner-city lead clinics is not always reliable in follow-up and therefore a lag time of two weeks leads to substantial numbers of children in need of rapid follow-up and in whom such cannot be achieved.

Recent observation that zinc protoporphyrin (ZnPP) can be measured in blood without need for an extraction procedure led to the development of instruments which read out directly in units of protoporphyrin in whole blood.[5-7] Such an instrument has the advantages of portability, immediate results, and requiring a small volume of blood. The reliability of the hematofluorometers and of the precision of FEP determinations has been questioned and this study was undertaken to clarify the usefulness of the hematofluorometer.

METHOD

Free Erythrocyte Protoporphyrins (FEP)—At the time of a patient visit, venous blood was drawn and divided into four aliquots. Each aliquot was assigned a labeling character so that later comparisons could be made on *identical* blood specimens. Aliquots were sent to two laboratories (#1 and #2) each of which used the Piomelli single-extraction method for analysis.[1] ZnPP levels were obtained immediately on each of the two portable machines, (A and B). Both machines were standardized with the manufacturers' standard slides and recalibrated after every fifth sample was run.

Lead—Laboratories #1 and #2 performed whole blood lead determinations by atomic absorption spectroscopy on the aliquots sent to them.

RESULTS

Samples of blood from 144 patients were used in the investigation. It took 195 venoclyses, each divided into aliquots, to obtain complete data on 129. The problems encountered included broken specimen tubes, tubes lost in transit, and insufficient sampling for complete determination. All of these are directly referable to problems of utilization of a central lead laboratory. On no occasion did a hematofluorometer malfunction or fail to function.

Table 1 lists the values obtained on the two hematofluorometers and by the two laboratories. We have

*Dr. Marcus is Assistant Director and Dr. Ziering is a staff member, Department of Pediatrics, Newark Beth Israel Medical Center, Newark, New Jersey 07112. Correspondence may be addressed to Dr. Marcus at the Medical Center.

Table 1
Distribution of FEP Results According to CDC Risk Categories

	Extractian Procedure Results (μg/dl)		Hematofluorometer Readings (μg/dl)	
	Lab #1	Lob #2	Fluarometer B	Fluorometer A
30 μg/dl	31	44	27	7
30-60	41	40	35	38
61-190	57	41	59	63
190	6	4	8	21

Toble 2
Correlation coefficients comparing FEP determinations by Laboratories 1 and 2 and by hematofluorometers manufactured by A and B

	Protoporphyrin Results			
Pratoporphyrin Reparts	Lab #1	Lab #2	Fluorometer B	Fluorometer A
1	–	0.94	0.89	0.90
2	0.94	–	0.82	0.85
B	0.89	0.82	–	0.81
A	0.90	0.85	0.81	–

chosen to list them in the ranges selected because these are in the ranges in the "risk" categories suggested by the CDC.[8] Although it appears that there is a disproportionately greater incidence of higher FEP levels reported by Laboratory #1, there appears to be no significant difference when analyzed by the one-way analysis of variance tests.

Correlation coefficients were obtained comparing the results of every possible pair of determinations. These appear in Table 2. The coefficients suggest that there is a linear relationship between all of the "methods" of erythrocyte protoporphyrin determinations.

The whole blood lead determinations show a poorer correlation (0.75) than that of even the least well correlated EP measurements (0.81). Although again this was not significantly lower statistically, it raises once again a serious question as to the reproducibility of blood lead determinations.

Table 3 lists representative values obtained from various samplings. What is most striking is the wide range in the EP values compared to the variation in lead levels. This despite the fact that a more linear relationship exists between various EP measurements.

Toble 3
Examples of Values by Sample Number

Somple	FEP				Leod	
#	1	2	B	A	B	A
1	20	22	40	20	30	26
2	106	92	225	150	38	44
3	22	22	45	25	40	43
4	116	79	220	170	40	41
5	42	44	70	40	33	31
6	49	32	90	62	30	23
7	32	29	70	45	30	41
8	76	50	140	100	30	38
9	49	62	40	30	15	16
10	30	31	50	30	37	38
119	31	21	50	55	49	56
120	44	40	80	65	35	43
121	53	40	100	60	53	48
122	81	82	110	100	60	55
123	21	13	50	30	50	42
124	79	48	160	140	58	48
125	112	113	35	140	40	42
126	99	77	225	160	48	44
127	71	52	98	70	48	40
128	39	52	85	55	50	52
129	58	68	130	110	41	41

DISCUSSION

A rapid, reliable determination of the degree of lead poisoning can be of considerable value in any lead poisoning treatment program. It allows instant feedback to parents thus assuring personal satisfaction for bringing a child in for testing and fosters a feeling that the parent is part of the treatment "team."

The remarkable variation in results from the individual laboratories is as yet unexplained. Both laboratories cooperate with the CDC proficiency studies and have been shown to perform well.

Since making one hematofluorometer operational in our clinic, two children were admitted solely because of high EP levels. One had neither anemia nor x-ray evidence of lead, and was in fact asymptomatic. He was started on chelation two days later. His lead level was reported as 86 μg/dl. The second case had confirmatory basophilic stippling and positive x-rays and had a lead level of 146 μg/dl. It is clear that an unnecessary delay would have been imposed between samplings and institution of therapy in both cases. It is unclear what further damage might have occurred in the interim.

CONCLUSION

It appears that the hematofluorometer is a useful addition to a lead diagnosis and treatment program. It satisfies the need for a quick, portable, reliable determination of the biological effects of lead.

Acknowledgement—The two hematofluorometers were provided by the AVIV Company of Lakehurst, New Jersey and the Bromberg Company of Boston. We would like to thank the personnel of the Pediatric Clinic, Newark Beth Israel Medical Center, the laboratory of the New Jersey State Health Department, the Heavy Metals Laboratory of the Department of Preventive Medicine, CMDNJ. We would like also to thank Dr. Marvin Lavenhar, Department of Preventive Medicine, CMDNJ, for his guidance in the statistical analysis of our data.

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"Where or When": Still Mystery and Magic in the Guise of "Déjà Vu"

STEVEN R. KOHN, M.D., Hackensack†

"It seems we stood and talked like this before. We looked at each other in the same way then, But I can't remember where or when.
"The clothes you're wearing are the clothes you wore. The smile you are smiling you were smiling then, But I can't remember where or when.—
"Some things that happen for the first time,—Seem to be happening again.—And so it seems that we have met before, and laughed before, and loved before, But who knows where or when!"*

What sort of a phenomenon was Lorenz Hart thinking of in 1937 when he wrote those still-popular verses for the Rodgers and Hart production *Babes in Arms?* Was Hart hitting the single note of an emotion or an illusion unique to himself or to the poets, artists, or young lovers of his era or was he, in reality, striking an harmonious chord in the lives of millions of people from all fields of endeavor, in all epochs, and of all ages who had experienced at one time or another a strange and wonderful feeling that ". . . some things that happen for the first time,—Seem to be happening again"?*

Hart's "Where or When,"* perhaps unwittingly, was striking that common chord, and his sensation was the same as that experienced by a young pharmacist apprentice from Cheyenne whom I recently encountered. He was gazing in a perplexed manner at a selection of spring apparel in the window of a Greenwich Village haberdashery, and, while laughing somewhat nervously when I inquired as to the cause of his apparent consternation and confusion, replied that he was on his first visit to New York City and until two days ago had not been farther east than Davenport, Iowa. Speaking softly and hesitating, as though we were sharing a secret, he explained the problem. While standing in front of this men's shop on 8th Street at 11 a.m. on a damp and drizzly Saturday morning in January—that is, standing in front of a store he'd never visited, on a street he'd never walked, in a city which was totally strange and new—he suddenly was overwhelmed by the incomprehensible sensation that on some previous and far-distant occasion he had stood on just such a street and had gazed at just such an arrangement of cashmere jackets, tweed suits, and striped shirts. And, on that first occasion too, he had felt the damp of the drizzle on the back of his neck and heard the distressing battle sounds of mid-morning traffic. But, he continued, it was not just the feeling of oppressive and impossible familiarity that had him so perplexed, but also, and perhaps to a greater degree, the uncanny knowledge, which had just as swiftly taken hold of him, as had the familiarity-feeling, that just such a stranger as myself would pause on his passage down 8th Street and engage him in the sort of conversation we had just had.

"As when with downcast eyes we muse and brood,
And ebb into a former life, or seem
To lapse far back in some confused dream
To states of mystical similitude,
If one but speaks or hems or stirs his chair,
Ever the wonder waxeth more and more,
So that we say, 'All this hath been before,
All this has been I know not when or where.' "

Tennyson, *Early Sonnets*

*"Where or When" from *Babes in Arms* by Richard Rodgers and Lorenz Hart. Copyright 1937, Chappell and Co., Inc.

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WHAT IS THE PHENOMENON?

What then can such a phenomenon be called, how often and to whom does it occur, and, further, how may such a mystery be explained?

In the mid-19th century, French neurologists and psychologists, such as Falret, Voisin, and Herpin, placed this sensation in the broad category of the "aura intellectuale." Later, such English investigators as the famed Hughlings Jackson relegated the phenomenon to the magical-sounding group of "dreamy mental states," and often referred to it as "mental diplopia" or a "double consciousness." Freud considered the feeling "uncanny" and categorized it with his "fausse reconnaissance" or errors of recognition. At present, although the above groupings and descriptive terms still may be appropriate, the phenomenon has come to stand independently and is commonly known as the "déjà vu."

"Déjà vu," though its exact significance remains obscure, has acquired a precise psychological denotation, as well as a literal definition and several rather loose literary connotations. As a form of paramnesia or defect of memory the "déjà vu" signifies "the illusion of remembering scenes and events experienced for the first time." As in Hart's "Where or When"* or in the problem of the young man from Wyoming, an individual experiences "déjà vu" when, upon meeting a strange person or a previously unknown set of circumstances, he feels suddenly and quite certainly, on one hand, that this strange person or strange situation has been encountered some time in the past, while, on the other hand, he knows with as great a certainty that such a previous encounter never really occurred. "Déjà vu," then, is *an inappropriate feeling of familiarity with new events or with new surroundings.*

Such a deep and comprehensive psychological definition as presented above must not be confused with the literal definition of "déjà vu" used by magazines or newspapers. *Time,* for example, said of the 1965 New York World's Fair media ". . . have made virtually all the offerings of science, architecture, and foreign culture 'déjà vu'." Another recent statement was that Philippe de Gaulle, ". . . is a sort of 'déja vu' copy of his father. . . ." In these examples "déjà vu" signifies that which has been "already seen." Random House defines that which is "déjà vu" as "unoriginal, as a trite story situation or a dated film technique."

DOES EVERYONE EXPERIENCE "DÉJÀ VU?"

"*Everybody* is familiar with those
strange sensations we sometimes have
that our life for the moment exists
in duplicate, that we have lived
through the moment before or shall again."

Thomas Hardy, *A Pair of Blue Eyes*

Is *everybody* familiar with those strange sensations? Has everyone experienced "déjà vu?" Early investigators of the phenomenon, such as André Lalande in 1893 and J.T. MacCurdy a quarter-century later, estimated that 30 percent of the adult population had experienced "déjà vu," while W.H. Burnham guessed, in 1889, that closer to 50 percent of the population had had the experience.

Not satisfied with these results, A.H. Chapman and Ivan N. Mensh reported a detailed study of "déjà vu" and conscious fantasy in adults in the *Psychiatric Quarterly—Supplement* in 1951. Their careful survey indicated that 35 percent of women and 31 percent of men recalled "déjà vu" experience(s). "Déjà vu" had an especially high incidence among professional, student, and clerical groups and a low incidence among non-travelers. The highest incidence was in the twenty to thirty-five age range and the greatest frequency appeared in adolescence and early adulthood. Age, they concluded, is the most significant factor with regard to incidence and frequency of "déjà vu."

Interestingly, though no upper limit has been placed on the age at which an individual may recall having experienced "déjà vu," there appears to be a limit at the lower end of the time scale. As Sir James Crichton-Browne put it, "The fact is that they [déjà vu"] emerge only at a certain state of mental evolution, rarely occurring in a child under eight years of age. . . ."

Reviewing the world's literature on the subject, this writer has found no record of a "déjà vu" experience before the age of seven, except for those of philosopher P.D. Ouspensky at age six. It seems, moreover, that the people who recall having "déjà vu" experiences in childhood most frequently chose eight as the earliest age at which the experience can be recalled.

Walker Percy put the adolescent experience of the phenomenon in interesting perspective when he wrote of Williston Barrett in his 1966 novel *The Last Gentleman:* "As a child he had had 'spells," occurrences which were nameless and not to be thought of, let alone mentioned, and which he therefore thought of as lying at the secret and somehow shameful heart of childhood itself. . . . It was 'déjà vu'. . . ."

HOW CAN "DEJA VU" BE EXPLAINED?

"Yossarian shook his head and explained that 'déjà vu' was just a momentary infinitesimal lag in the operation of two coactive sensory nerve centers that commonly functioned simultaneously"

Joseph Heller, *Catch-22*

How can a phenomenon which seems as mystical and magical as does "déjà vu" be explained? Can it be understood neurologically, psychiatrically, psychologically, or philosophically? Is it comprehensible in the terms of all these intellectual spheres or not in any? What do you tell Lorenz Hart's lovers? How do you explain the mystery to a confused visitor to a strange city or to such notables as General George S. Patton, William Hone, Lord Lindsay, John Buchan, and Marcel Proust who also had been troubled by the mystery? Perhaps you can reply with an empty answer as Jachid did to Jechidah's questions, during their very first meeting, in a story from Isaac Bashevis Singer's 1964 collection *Short Friday:*

". . . Jechidah said: 'I have a strange feeling I have experienced all this before.'
" 'Déjà vu'—that's what psychology calls it.'
" 'But maybe there's some truth to it. . .'
" 'What do you mean?'
" 'Maybe we've known each other in some other world.'
"Jachid burst out laughing. 'In what world? There is only one, ours, the earth.'
" 'But maybe souls do exist.'
" 'Impossible. What you call the soul is nothing but vibrations of matter, the product of the nervous system. I should know, I'm a medical student.' "

Perhaps Yossarian's answer to the chaplain is the one that should be given, though novelist Joseph Heller has said privately, ". . . there is really no scientific basis to my explanation I made it up" A quick look into the neurological literature might suggest, however, that Heller accidentally expressed the best scientific explanation of "déjà vu" brought forth to date. In 1963, *Brain* published Robert Efron's brilliant paper, "Temporal Perception, Aphasia and Déjà Vu." Specifically, Efron said that an individual must be able both to characterize (identify) an event and determine the occurrence of the particular event in relation to other events in order to act effectively and to deal appropriately with the events in his environment. There must be, then, both "information" and "sequence" in order for an event or a situation to have "meaning." The human brain is divided into a right and a left hemisphere, and the left half of the brain is considered "dominant" for speech in right-handed individuals as well as in the majority of left-handed individ-

uals, while the other hemisphere is "nondominant." Keeping this in mind, Efron's hypothesis is in part ". . . that certain information (relative to the time of occurrence) is transferred from nondominant to dominant hemisphere before temporal lobe discrimination of simultaneity and order can be performed."

Hypothetically, this transfer of information from nondominant to dominant hemisphere normally requires an undetected delay of a few thousandths of a second (milliseconds). The environment actually presents both hemispheres with the same information simultaneously. But, if the normal delay in transmission from nondominant to dominant hemisphere, as Efron proposed, were extended for several hundred milliseconds, the dominant hemisphere would of necessity receive essentially the same information twice, directly from the environment and again after a delayed relay from the nondominant hemisphere. Or, as Yossarian explained, there "was just a momentary infinitesimal lag in the operation of two coactive sensory nerve centers that commonly functioned simultaneously."

From these events the "déjà vu" phenomenon follows naturally. Under these circumstances a person might think, "Everything which I am perceiving *now* already has occurred," because it already had been "logged-in" by the dominant hemisphere via the direct pathway or "Everything is happening twice." Such an idea is unacceptable to a human being who would rationalize it away with the old familiarity feeling of "déjà vu." P.D. Ouspensky's literary hero Ivan Osokin cried out against such a thought: "That alone is enough to drive one mad."

Efron's theory also serves to explain the close association of "déjà vu" with clairvoyance or, as the young man from Wyoming explained that he was perplexed by this feeling of familiarity in a strange situation, but he was even more upset by the foreknowledge of our chance meeting and the facile predictability of the exact words of our conversation. Clairvoyance, explained Efron, occurs because the individual experiencing the "déjà vu" has more information at his disposal after the previously described state of delayed interhemispheric transmission has persisted for a few seconds than he had earlier. "He can then say 'I am aware that a series of events are [*sic*] apparently occurring twice. As this is unacceptable . . . the more likely alternative is that I am merely anticipating what will occur'." In other words he refers to clairvoyance.

"GLIMPSES OF FORGOTTEN DREAMS"?

Moreover, something is or seems
That touches me with mystic gleams,
Like glimpses of forgotten dreams—
Of something felt, like something here;
Of something done, I know not where;
Such as no language may declare.

Tennyson, *The Two Voices*

Although Efron's proposed explanation of "déjà vu" is more than adequate and seems generally to have been borne out by the experiments of Wilder Penfield and his co-workers in Canada, it is only one of more than a dozen provocative concepts which, in an attempt to disprove Tennyson's suggestion that "déjà vu" is a phenomenon whose nature is "Such as no language may declare," cover a broad spectrum of thought and speculation. In psychoanalytic terms, "déjà vu" may be the result of a previously experienced fantasy which, at the time of the feeling of "déjà vu" is being experienced in reality. In 1904 Joseph Grasset described a variant of this fantasy concept in the *Journal de Psychologie* when he explained that, to him, "déjà vu" was the result of "a subconscious impression previously received, but only reaching consciousness under the influence of a new similar impression." The following year Freud explained "déjà vu" in terms of another variant of the original fantasy concept when he said in his *Psychopathology of Everyday Life* that "déjà vu" ". . . corresponds to the memory of an unconscious fantasy." By this Freud implied that ". . . in such moments something is really touched that we have already experienced, only we cannot consciously recall the latter [event] because it never was conscious." To illustrate his point, Freud analyzed one case of "déjà vu" in which he thought the feeling was persistent and particularly intense:

"A woman of thirty-seven years asserted that she most distinctly remembered that at the age of twelve and a half she paid her first visit to some friends in the country, and as she entered the garden she immediately had the feeling of having been there before. This feeling was repeated as she went through the living-rooms, so that she believed she knew beforehand how big the next room was

When she decided upon this visit she knew that these girls had an only brother, who was seriously ill. . . . and thought to herself that he soon would die. But it happened that her own only brother had had a serious attack of diphtheria some months before, and during his illness she had lived for weeks with relatives far from her parental home. She believed that her brother was taking part in this visit to the country. . . .

. . . the expectation of the brother's death had played a great part in the girl's mind at that time, and . . . either it never became conscious or it was more energetically repressed after the favorable issue of the illness She found the analogous situation in her friends' home; their only brother was in danger of an early death, an event that really came to pass. . . . She consciously might have remembered that she had lived through a similar situation a few months previous, but instead of recalling what was inhibited through repression she transferred the memory feeling to the locality, to the garden, and the house, and merged it into the *fausse reconnaissance* that she had already seen everything exactly as it was."

Freud concluded that, from the fact of the repression, the expectation of the death of his patient's brother was most likely wish fantasy and may well have been the original provoking agent for the "déjà vu."

Freud's analytic views are now part of the great body of ego defense mechanisms in which the role of "déjà vu" is thought by people such as C.P. Oberndorf, Jacob Arlow, and Louis Linn to be that of a mental mechanism through which a familiarity feeling causes an individual to feel secure and comfortable in dangerous or unpleasant situations. A person might say to himself, as Oberndorf suggested, "Don't worry about this frightening predicament. You have been in this spot before and escaped unharmed." Such a "déjà vu" experience might well occur to a nervous visitor to a strange city or to a war-weary soldier on his way to another battle. As Leslie A. Fiedler put it literarily, ". . . 'déjà vu' takes the curse off the whole ticklish enterprise. . . ."

Eli Marcovitz and others suggested that "déjà vu" may be ego defensive when it is in the form of an "encore vu." That is, an individual may feel that he is repeating an unsatisfactory experience in an effort or desire to transform it into a satisfactory one. Here "déjà vu" functions to help one say: "I am experiencing all of this again; I am having a second chance."

R.W. Pickford and J.T. MacCurdy of Glasgow saw the importance of "déjà vu" as a defensive method for rationalizing paramnesias. In this case a "strange" mental intrusion perplexes the individual. In an effort to rid himself of this unpleasurable and undesired thought, he uses "déjà vu" to transform it into something "familiar," and possibly even pleasurable.

Following other leads, such as Tennyson's ". . . glimpses of forgotten dreams" (1833), such early researchers as Buccola in 1883, Sully in 1884, and Sándor Ferenczi in 1905 suggested that "déjà vu" experiences were the result of a previous night dream or daydream whose content at the time

of the "déjà vu" was being experienced in reality. Of such fantasy material, Sully said, they are ". . . unauthorized mints which issue a spurious mental coinage, and so confuse the genuine currency." One may correlate such ideas with the more recent ones of Dr. Bernard Zuger (1966) and suggest that whereas dreams often consist of the residual material from daily mental activity while awake, "déjà vu" may arise from dream material. Thus the dream and the "déjà vu" are part of a single phenomenon called "dream" under some circumstances and "déjà vu" under others.

E.B. Titchener, Emminghaus, Théodule Ribot, and others saw "déjà vu" not as the result of fantasy material, but rather as the result of "a moment of distraction between two perceptions of the same place," this idea sounds like the psychological wording of Efron's neurological hypothesis. Such a "moment of distraction" may occur during a moment of abnormal weakness of mental associative tendencies. For example, a person who is about to cross New York's busy 42nd Street looks hastily east and then west, automatically assessing traffic; now his attention is momentarily held by a display in a store window and he pauses for a few seconds before actually crossing the street. As he crosses the street he thinks "Why I've crossed the street already just a few moments ago," for "déjà vu" occurred when the "looking across" was briefly disjoined from the consciousness of the "walking across."

In 1911 Havelock Ellis claimed that "déjà vu" resulted from the inexact recollection of an actual previously experienced situation which is similar to the present situation or, more imaginatively, from "a reversed hallucination" in which the individual accepts the reality of the present moment as if it were merely a representation or an illusion of reality and consciousness interprets the enfeebled representation as if it were merely an old memory or "déjà vu."

Plato and Plotinus, as well as members of certain extant religious and parapsychological groups have considered "déjà vu" both the result of and serious evidence for the phenomenon of reincarnation. If the soul is immortal, man comes into life with knowledge retained from previous existences, and, when he experiences "déja vu," he is actually recalling the same event as it occurred in a previous incarnation. Such a concept must have represented a source for a final explanation for the "déjà vu." F.W.H. Myers in a poetically pleasing, but as yet rationally unfounded idea regarded "déjà vu" as the recollection of ancestral and antenatal memories hereditarily transmitted as fantasies, images, and other mental phenomena.

CONCLUSION

"Where or When"* remains in Evan Hunter's words "mystery and magic in the guise of 'déjà vu'." Were one to attempt an explanation of "déjà vu" based on any one of the above causes, reasons, or mechanisms none of which has been proved or disproved, one still would be left with a phenomenon whose very nature seems perpetually to cry out from the deepest recesses of all that is unknown in man's mind and soul.

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Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

INDICATIONS: *Therapeutically,* (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: infected burns, skin grafts, surgical incisions, otitis externa; primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia); secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis); traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically,* the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

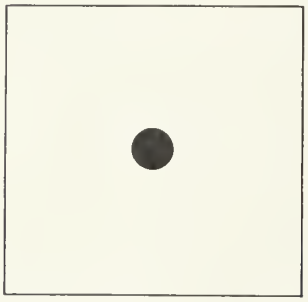
When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

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Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

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***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

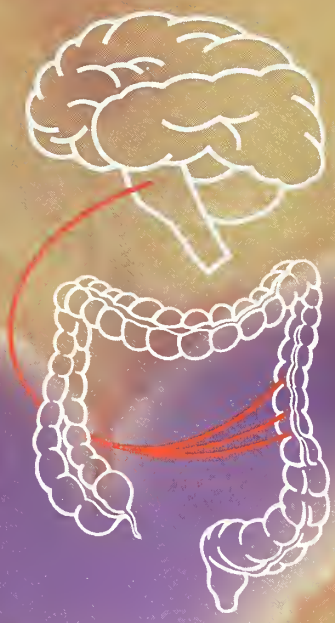
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Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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WE'RE FIGHTING FOR YOUR LIFE

Regulations, Licensure, Medical Discipline, and Self-Regulation of Physicians

JOSEPH A. RIGGS, M.D., Haddonfield

The Medical Society of New Jersey should be congratulated for planning a conference regarding the regulation of physicians and their competency. The public challenge now implies that objective evidence of continuing competence must be determined periodically. The history of the New Jersey State Board of Medical Examiners, along with the attitudes and functions of the Board, and the composition and activities of the present Board, are presented. Problems of the incompetent physicians are discussed. These problems may be solved by prevention, identification, and action. Suggestions are made as to how to help the incompetent physician.

The Medical Society of New Jersey should be congratulated for having the wisdom to plan a conference about medical discipline, which is indeed quite necessary and long overdue. The medical profession increasingly is being challenged to provide assurance to the public that individual practitioners are, in fact, preserving and extending their professional competence. Although the challenges are annoying to the vast majority of physicians, who continually are committed to improving their competence, they often are justified in many ways.

"Up until very recently, the issuance of a license to practice medicine and the award of a specialty board certificate were single episodes, occurring early in a physician's career. It was presumed that all physicians having demonstrated their degree of competence by receiving a license and also, in many instances, a board certificate would continue to exhibit the same degree of competence throughout their practicing careers. The public challenge now implies that this is not necessarily true and that objective evidence of continuing competence must be determined periodically, and possibly should be *required* periodically of all physicians. The Federal and State Governments, the State Medical Board, the Federation of State Medical Boards, the American Board of Medical Specialties, and several medical specialty societies, have, within the past few years, addressed the subject of relicensure and recertification, as well as the very broad subject of continuing medical education."[1]

My purpose, as a representative of the New Jersey State Board of Medical Examiners, is to present some concepts concerning the Board's functions, and then to outline some facts and figures regarding the activities of the Board.

First, let me define the attitude of the Board:

"The Board recognizes that to practice medicine is not a natural right of any person, but it is a privilege granted by legislative authority, and that it is necessary in the interest of the *health, safety,* and *welfare* of the people of the State [of New Jersey] to provide for the granting of that privilege and the regulation of its use. The Board demands that the public be protected from the practice of medicine by *un*authorized or *un*qualified persons and from *un*professional conduct by persons who are already licensed to practice medicine"[2] [in New Jersey].

STRUCTURE AND FUNCTIONS OF THE BOARD

The New Jersey State Board of Medical Examiners is in the New Jersey Department of Law and Public Safety, under the Division of Consumer Affairs, and is one of the nineteen professional boards in the State of New Jersey. The Board

*Based on a presentation before the Presidents and Presidents-Elect of County Medical Societies and Specialty Societies, Chiefs of Staffs of New Jersey Hospitals, and the Board of Trustees of MSNJ, April 13, 1978, North Brunswick. Dr. Riggs is President, New Jersey Obstetrical and Gynecological Society and a member of the New Jersey State Board of Medical Examiners. Correspondence may be addressed to Dr. Riggs at 44 Tanner Street, Haddonfield 08033.

was established by legislation on May 22, 1894, and is responsible for the regulation of the practice of medicine in the State of New Jersey.

The Board examines, licenses, and registers all physicians who practice in the State of New Jersey. It holds disciplinary hearings when necessary, enforces the administrative rules and regulations of the Board, and investigates violations of the Medical Practice Act by lay people or other individuals not authorized to practice medicine in New Jersey.

In addition, eight other categories of professional licensure are under its jurisdiction, namely:

1. Podiatry
2. Chiropractic
3. Physical Therapy
4. Physical Therapy Assistants
5. Laboratory Directors
6. Midwives
7. Hearing-Aid Dispensers
8. Orthoptics

In the practice of medicine, the Board licenses both medical and osteopathic doctors. The Board procedure for licensure is thorough and comprehensive. All licensees are compelled to submit a formal application supplied by the Board and clearly must demonstrate their graduation from a professional school acceptable to the Board. They also must demonstrate postgraduate training as required by State law. All applications are reviewed thoroughly before licensure. In addition, all applications are checked against a master file card system which reflects any Board disciplinary action against an applicant in another State. Any action taken against a physician in another State is reported through the Federation of State Medical Boards and the New Jersey Board receives these reports. If that licensee from another State is also licensed in the State of New Jersey, appropriate documentation of the disciplinary action is acquired. This then is presented before the New Jersey State Board for appropriate action against the physician's New Jersey license. As you can see, there is a good system of communication between the sister states.

The activities of the New Jersey State Board of Medical Examiners, prior to 1973, were quite limited. Commencing in 1973, a joint effort has been under way to improve the proficiency of the New Jersey Board. It seems that the increased activities of the Board are due to:

1. The pressures of consumerism.
2. The hiring of a full-time executive secretary in early 1973 to coordinate and administer the activities of the Board.
3. The cooperation and excellent work done by the deputy attorney generals assigned to the Board of Medical Examiners.
4. The exhaustive investigative work conducted by the Enforcement Bureau.
5. The conscientious and diligent manner that the Board has demonstrated in regulating the practice of medicine.

The Board staff has increased from a few secretaries in a small office in 1972, to, at present, a full-time Executive Secretary, a full-time Assistant Executive Secretary and a staff of 10 secretaries, in a larger office measuring 2,200 feet. Mr. Alfred Schuster is the Executive Secretary and his services have been invaluable in coordinating the activities of the Board. He was instrumental in enforcing registration of all physicians so that the Board is now able to tell just how many physicians there are, where they are located, in what specialties they are practicing, and so on.

The deputy attorney generals have been increased in the past five years from one part-time deputy, who had jurisdiction over 19 Boards, to three full-time deputies for the Medical Board alone. These deputies are involved as legal counsel to the Board and formulate, at the direction of the Board, appropriate orders relative to licenses. In addition, they try many cases before a hearing officer or before the Board.

New Jersey also has an excellent Enforcement Bureau, which conducts both open and undercover investigations and submits detailed and informative written reports to the Board. The Board attempts to take immediate action based on the information presented.

Who are the individuals who constitute the New Jersey State Board of Medical Examiners? The Board is composed of the following officers and members:

1. Edwin A. Albano, M.D., President
2. Jordon D. Burke, M.D., Secretary
3. Rudolph T. DePersia, M.D., Treasurer

There are 16 voting members of the Board, including:

1. Nine doctors of medicine
2. One doctor of osteopathy
3. One chiropractor
4. One podiatrist
5. One laboratory director
6. Two consumers
7. One governmental member

Not included are the three deputy attorney generals, who act as legal counsel, the Chief Inspector of the Enforcement Bureau, who verifies the information submitted and the Executive Secretary, who coordinates the agenda and activities for the Board. These persons do not vote, but rather assist the Board in its decision making. This is a conscientious and diligent Board, consumer oriented, and willing to spend as many hours as necessary to regulate the practice of medicine and supervise the conduct of licensees. Quick and decisive action is taken by the Board in most disciplinary cases. In addition, the Board establishes policy and outlines rules, regulations, and guidelines directed to assure the health, safety, and welfare of the public.

"From the consumers' point of view, the Board of Medical Examiners' purpose is to protect them from 'bad' doctors. But doctors are human, and human beings are rarely *all bad* or *all good.* The Board finds that most physicians are very responsible persons. However, some try to be too 'good' in too many directions at one time. The results can be a terrible mess. For example, sometimes a hard-driving physician enters practice and he strives to get more and more patients. In just a few years, the result is a lack of time: too little time for sleep; too little time for exercise; too little time for his family. Eventually, the mental and physical strains become so great that the physician cannot deal with them. Instead, he may turn to alcohol to relieve tension, or he may use stimulants to keep him going longer hours, or later he may even use sedatives to sleep. Finally, he also may get into trouble with drugs, which he begins to take because of this chronic never-ending pressure. The best term for such a situation is irresponsible. Oftentimes this results in irresponsible behavior toward patients, but beyond that, there lies a deeper irresponsibility. This basic irresponsibility is a lack of this physician's attention to himself. If the physician takes good care of himself, he is apt to maintain his judgment intact. If his judgment is intact, he is apt to treat himself, his patients, and his family well."[3]

The Board protects the people of the State of New Jersey by identifying deviate medical behavior early, and attempts to foster positive action, treatment, and rehabilitation for those physicians involved. It has been said that "the effectiveness of a State Board of Medical Examiners should be

judged by the number of physicians it is able to salvage, rather than by the number of licenses it lifts."[2] For many years, "the known national annual rate of loss of physicians from practice of medicine in the United States as a direct result of some form of deviate behavior has exceeded seven percent of the number graduating from the country's medical schools each year."[3] There are approximately 118 medical schools in the United States, each with an average graduating class of 110 physicians. That makes approximately 13,000 physicians graduating each year. Of those graduating, more than 100 physicians will be lost to suicide, 250 will become ineffective through the use of drugs, and another 500 through the use of alcohol.

During the years 1974 through 1977, the Board of Medical Examiners met an average of three times a month resulting in the following statistics:

1. There were 431 appearances before the Board.
2. There were 77 full hearings held.
3. There were 86 licenses that were either suspended, revoked, or voluntarily surrendered.
4. There were eight revocations of registration certificates for Controlled Dangerous Substances.
5. There were 522 penalties assessed.
6. The amount of the penalties assessed, including cost equalled $972,822.

In addition to disciplinary actions the licensing activity for physicians to practice medicine in the State of New Jersey has been increasing rapidly in recent years (Table). For example, during the year 1960, there were 576 physicians who were licensed. In 1970, this figure increased to 976. In 1977, there were 1,620 physicians licensed to practice. In the past four years, there have been 6,055 licenses granted in the State of New Jersey, which represents approximately 25 percent of the existing physicians in the State of New Jersey.

There has been an organized movement in political circles in Washington, D.C. that medical licensure should be a *federal* concern. Many consumer groups, some possessing physician-spokesmen, have supported the Federal Government in this regard. However, the New Jersey State Board of Medical Examiners has demonstrated that licensing and regulation of the practice of medicine can be controlled completely, in an excellent and efficient manner, by individual *state* boards of medical examiners, which are not under federal control. The New Jersey Board is gaining a nationwide reputation as a Board that is dedicated to the service of the people of its state and which takes immediate action against physicians who drift from the recognized standards of practice or who violate New Jersey State Law regulating the practice of medicine. In maintaining this posture, the Board, on many occasions, has been subject to criticism and complaints from members of the medical profession. There apparently has been considerable misunderstanding by those who do not have a working knowledge of the activities of the State Board. Much of the criticism of the Board's operations comes from those who have had neither direct involvement nor factual understanding of how a medical board conducts the business of protecting the people of the State of New Jersey. It is unfortunate and distressing that the opinions of these uninformed individuals are being quoted in the news media as *fact,* with very little input from the Board itself.

Table 1
Total New Jersey Licensed Physicians on a Yearly Basis (M.D.'S and D.O.'S)

Year	Total	
1960	576	
1960	566	
1962	533	
1963	555	
1964	524	
1965	531	
1966	557	
1967	583	
1968	766	
1969	904	
1970	976	
1971	1,081	
1972	1,145	
1973	1,511	
1974	1,401	6,055
1975	1,430	
1976	1,604	
1977	1,620	

THE INCOMPETENT PHYSICIAN

The Board is being pressured to do something about the incompetent physician. What can be done? The answer to that question should be quite simple, because it is well outlined in the AMA Principles of Medical Ethics, which states of physicians: "They should expose, without hesitation, illegal or unethical conduct of fellow members of the profession."[4] However, the answer to this question is not simple, because it is quite difficult to define just what competency is. Is competence the same for an obstetrician as it is for a perinatologist? It could possibly be defined "by addressing the multiple components of the physician's professional activity, namely:

1. Knowledge.
2. The use of knowledge.
3. Technical skills.
4. Behavioral characteristics.
5. The process of care employed by the physician.
6. The outcomes of the patients cared for.
7. The ethical approach to practice.

Obviously, it becomes a Herculean task to determine all those components of the physician's activity."[2] So what do we do about the incompetent physician? One might possibly try to answer that question by three different approaches:

1. Prevention.
2. Identification.
3. Action.

"In regard to *prevention,* we must try to halt the influx of additional incompetent physicians. This is a shared responsibility by the educational institutions, by the accrediting bodies, by the licensing and certification bodies, and by the Federal establishment."[5]

1. The medical school must select a student who is qualified best to become a physician, and not select a student because that student is black, or Spanish, or a female, or has rich parents. The medical schools must assume the responsibility for awarding an M.D. degree only to individuals who have acquired the competence necessary to move to the next level of medical education and not award a degree based on a certain quota. They should also include a course in professional ethics (less than 60 percent of the medical schools teach such a course).
2. The State Boards play a major role in preventing incompetent physicians from entering practice by listing strict licensure requirements.
3. "The Federal Government through its Office of Immigration, must not perpetuate a program that invites any physi-

cian to immigrate without making certain that he or she is competent to enter the educational continuum, with essentially the same prospect of successful completion as graduates of American medical schools."[5]

Identification of the incompetent physician is a responsibility that is shared broadly among many:

1. Physician peers, by means of PSRO's tissue committees, audit committees, utilization committees, and morals and ethics committees can identify the incompetent physician better than anyone. However, too often they look the other way.
2. Professional organizations, such as the county societies, the Medical Society of New Jersey, and the specialty societies, can identify their problem physicians by the phone calls and letters of complaint received.
3. Hospitals can identify them by reports received from patients and from observations of other physicians regarding the incompetent activities of the professionals on their staffs.
4. Licensing and certifying agencies also can identify the incompetent physician by the nature of the deviant behavior noted.
5. Third party payers can identify incompetent physicians by the requests received for payments for the many questionable procedures done by the incompetent or unethical person.
6. Testing agencies, such as those required for recertification, or in some states for relicensure, also can "test-out" the incompetent physician.
7. Lawyers can identify the incompetent physician by multiple malpractice cases.
8. Consumers identify the incompetent physician by reporting deviant activities to the Judicial Council of the County Medical Society, or to the State Medical Society or the State Board of Medical Examiners.

Once the incompetent physician is identified, what *action* is suggested? This is the most trying obligation that we in the profession have. Nonetheless, the State Board, certifying agencies, the medical societies, the hospitals, the individual physicians, third party payers, and consumers *all* have a responsibility to assure that appropriate action is taken. Action can be taken by third party payers by withholding certification or denying recertification. Finally, it can be taken by the Medical Society of New Jersey by suspension from membership. Scaling down the list of sanctions from the least to the most serious, the following actions can be taken:

1. Reprimand.
2. Probation.
3. Suspension from organizational membership.
4. Ineligibility for third party payment.
5. Loss of certification or prevention of recertification.
6. Loss of hospital privileges.
7. Temporary or permanent loss of license.
8. Arrest.
9. Fine.
10. Jail.

There are some very important deterrents to appropriate action against the incompetent physician. The law itself, which protects the individual physician's constitutional right to practice, makes it very difficult to protect the public against the incompetent physician. Also, a major deterrent is the right of the accused physician to sue the reporting physician or lay person. Consequently, many states have amended Medical Practice Acts to protect reporting individuals with immunity from liability and the effects of such action have been enormous. For example, the State of Virginia has ordered that all incompetence must be reported to the State Board of Medical Examiners. "All physicians, hospital administrators, state officers, local medical societies, and state medical societies are required, by law, to report all instances of professional incompetence to the state licensing boards. They are also required to report any information indicating that a health professional is in need of treatment, or that he has been admitted to a health institution for the treatment of alcohol abuse, drug addiction, or psychiatric illness. They also must report information indicating that a health professional may be guilty of unethical, fraudulent, or unprofessional conduct. The Virginia law also states that any person making such a report is *immune* from any civil liability resulting therefrom, unless the reporting person acted in bad faith or with malicious intent."[6] After similar law was passed in Arizona, the number of complaints quadrupled within a short period of time.

Finally, we must not talk only in negative terms, that is, those actions resulting from disciplinary sanctions and deterrents. We also should look for incentive strategies to reward physicians for positive desirable behavior. There is a difference between controls and incentives. "Controls necessarily operate by establishing a *minimum* level which all providers must meet, and they supply no pressure to exceed the minimum. On the other hand, incentives operate on all providers all of the time, and encourage *maximum* attention to obtain improved results, even on the part of the very best physicians and hospitals. The pressure is thus for performance and not just for compliance."[7] Such incentives could possibly be in the medical liability insurance area, where it has been suggested that there should be incentives favoring excellent patient care by the awarding of decreasing premiums for those physicians who are competent and maintain a good record. Other incentives for optimal behavior of physicians may take the form of recognition by honor and award by local, state and national organizations. Other areas could be in the form of a non-coercive system of peer aid to disabled and incompetent physicians—call it "DAD" —Doctors Aiding Doctors. This peer review committee, to help the sick physician, could be available to pursue complaints and persuade the physician to seek help. They then can follow up to see the physician's response. Finally, the *last* suggestion might be a hotline phone number open 24 hours a day to hear problems and obtain information regarding an incompetent physician.

SUMMARY

It is evident today, that:

1. The public, i.e., the consumer, through its elective officials, is demanding that the physicians continue to perform effectively in the practice of medicine.
2. Continuing medical education is being emphasized and should be supported vigorously.
3. Many specialty boards are approaching the process of recertification and some are auditing patient records to provide assurance of daily competence in the care of patients.
4. PSRO now is attempting to evaulate patient care and is concerned with the reduction of the cost of patient care.
5. State Boards of Medical Examiners are becoming vigorous in their criteria for licensure and in 17 states they are demanding a specified number of hours of approved continuing medical education as a basis for relicensure.
6. The Federal Government progressively is enacting legislation that is trying to lead to a dominant and direct role in the private practice of medicine.

We must maintain the high standards of the practice of medicine, and accept the responsibility of identifying and disciplining the incompetent physician. This is a hard job, and we are faced with the responsibility of carrying out this job in the State of New Jersey.

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Sinus Histiocytosis with Massive Lymphadenopathy in an Adult

K. SARANATHAN, M.D.
MICHAEL A. NEVINS, M.D.
SOO JAE KIM, M.D.
EDWARD WAGMAN, M.D., Paramus

A 51-year-old black woman presented with typical clinical and pathologic features of sinus histiocytosis with massive cervical lymphadenopathy. Heretofore, this has been thought to be an unusual disease of children, but this benign disorder also should be considered in adults clinically presenting with suspected malignant lymphoma. Marked elevation of EBV antibody titers against capsular antigen and delayed transformation of lymphocytes challenged with mitogens were documented in this case. The etiology remains an enigma but may represent an acquired immunologic defect which makes susceptible individuals subject to abnormal proliferative responses after exposure to antigens such as virus infections.

In 1969, Rosai and Dorfman described a benign pseudo-lymphomatous disorder which they termed "sinus histiocytosis with massive lymphadenopathy" (SHML).[1] Subsequently, they reviewed 34 additional cases from throughout the world.[2]

The condition is characterized by cervical lymphadenopathy, fever, leukocytosis, anemia, and hypergammaglobulinemia. The pathologic picture is quite distinct with capsular and pericapsular fibrosis and sinuses packed with histiocytes showing marked lymphophagocytosis.

The present case is unusual in that it involved a 51-year-old female; previously this entity has been encountered almost exclusively in children. Immunologic studies revealed normal proportions of T and B cells but delayed response to phytohemagglutinin *in vitro*. Epstein-Barr virus (EBV) antibody titers against capsular antigen were elevated markedly initially and fell on subsequent evaluation. Although the pathogenesis is obscure, it has been suggested that SHML is the expression of an infection in an immunologically deficient individual.

CASE REPORT

A 51-year-old black female was hospitalized because of a three-week history of fever and extreme bilateral cervical lymphadenopathy. Except for weakness, anorexia, and tenderness of lymph nodes, she was otherwise asymptomatic. She had been receiving no medication and the previous medical history was unremarkable except for a hysterectomy in 1969 because of fibroids. For several years she had worked in a factory that manufactured cosmetics and had occupational exposure to a wide variety of presumably inert inhaled chemicals.

The pertinent physical findings were temperature of 102°F, massive bilateral cervical lymphadenopathy involving the long cervical chain, submandibular and supraclavicular glands which were matted and firm (Figure 1). There were no discharging sinuses. Other than for a small firm lymph node detected in the left axilla, there was no other lymphadenopathy. The liver and spleen were not enlarged and the examination was otherwise within normal limits.

Laboratory investigations included the following: hemoglobin 8.5 gm%, hematocrit 27%, WBC 14,900 with neutrophils 89%, band 3%, lymphocytes 8%, monocytes 5%, platelet count 547,000, reticulocyte count 0.8% and first hour sedimentation rate 126 mm. The SMA-12 was normal except for alkaline phosphatase 102 and SGOT 71. Total protein was 8.1 gm and immunoelectrophoresis revealed IgG 2,254 mg, IgA 439 mg, IgM 207 mgs. Alpha-1-antitrypsin and haptoglobin were elevated, Coomb's test (direct and indirect)

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Figure 1—Lateral view showing massive cervical lymphadenopathy

negative, stools for occult blood negative. Serum iron was 10 mg%, TIBC 174 mg%, saturation 6%. Bone marrow aspiration revealed hypocellularity with mild focal erythroid hyperplasia, plasmacytosis and increased iron stores. There were no malignant cells.

X-rays of chest and abdomen were not contributory except for slight cardiomegaly. Liver and spleen scans were within normal limits. Tuberculin and fungal skin tests were negative as were repeated blood and urine cultures.

Biopsy of a right cervical node was performed revealing a proliferative reaction which was sinusoidal in distribution (Figure 2). The sinuses were distended markedly by histiocytes which contained phagocytosed lymphocytes and erthyrocytes (Figure 3). Some of the histiocytes showed hyperchromatism and occasional mitotic figures. The capsule was thickened and fibrosed (Figure 4). Plasma cells were noted within the cords and in sinuses (Figure 5). There were no epitheloid granulomata and special stains for fungi and other microorganisms were negative. Microscopic specimens were reviewed by members of the pathology departments of three other hospitals who concurred with the pathologic diagnosis of sinus histiocytosis.

During her hospital course, the patient remained mildly febrile but received no specific treatment. Initially, she had complained of mild pain because of extreme lymphoid hyperplasia but this diminished as cervical swelling gradually subsided. Her general condition remained satisfactory and at the time of discharge after two weeks she resumed her normal activities. Unfortunately, two months later the patient moved to another area and was lost to follow-up.

DISCUSSION

The clinical and pathological features of this case resemble those of sinus histiocytosis with massive lymphadenopathy as originally described by Rosai and Dorfman. The great majority of cases reported in the medical literature have been

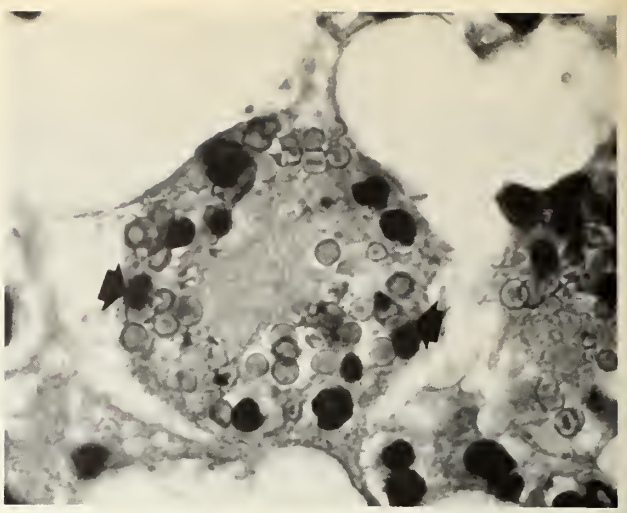


Figure 2—Marked dilation of lymph node sinuses occupied by many histiocytes and compressed inter-sinusal lymphoid tissue. (X 84)

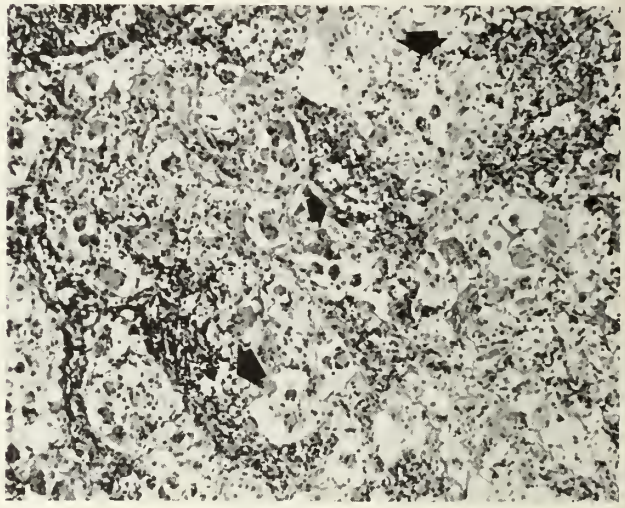


Figure 3—Erythrocytes, lymphocytes and neutrophils in a large sinus histiocyte.

in black children and only a few adults have been encountered with this clinical condition. In the first 34 cases reviewed by Rosai and Dorfman the disease appeared only in three after the age of 20 years (24,30, and 45 years respectively);[2] our patient apparently represents the oldest individual described with sinus histiocytosis. The clinical features were typical of SHML including massive cervical lymphadenopathy, fever, leukocytosis, elevated sedimentation rate, and hypergammaglobulinemia. The relatively benign clinical course with gradual spontaneous regression of lymphadenopathy is also consistent with this diagnosis. The only histopathologic feature that represented a departure from the classic description was the presence of an extreme degree of erythrophagocytosis which ordinarily is not described in this condition and might represent an element of malignant histiocytosis. However, there were no other features to suggest the latter diagnosis. Although histiocytes may acquire the potential for malignant proliferation, late transformation of cases with sinus histiocytosis to malignant histiocytosis has not been described to this time.

The etiology of sinus histiocytosis still is unsettled. Rosai and Dorfman postulated two possible mechanisms; a specific infectious process and an immune deficiency state. Attempts

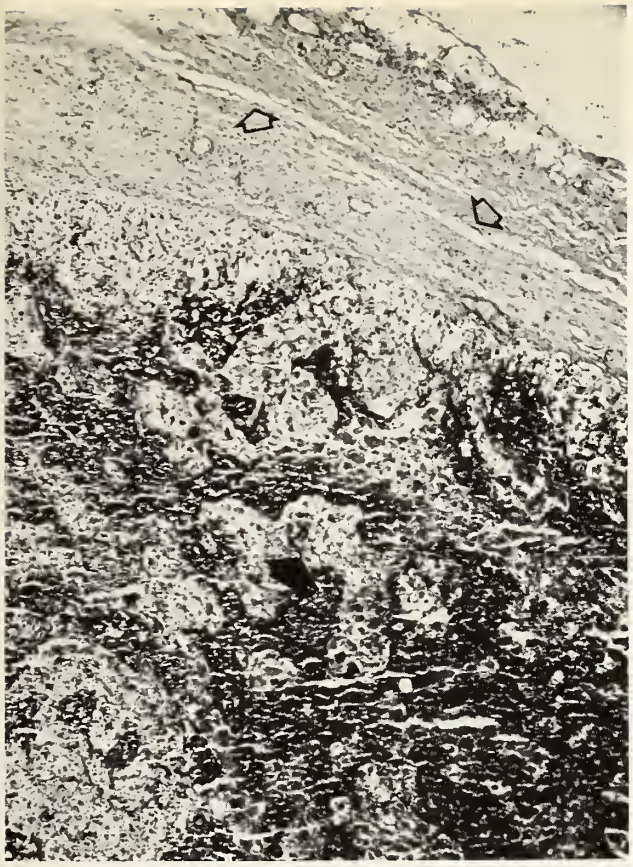


Figure 4—Cervical lymph node with capsular and pericapsular fibrosis, and dilated sinuses. (X 34)

at isolating an infectious agent have been unsuccessful but antibodies against Epstein-Barr virus have been identified in the serum of six patients with this disease, titers of 1:640 being reported in two. Lober *et al.* reported a case of SHML in a six-year-old black boy who had a titer of 1:320 against EBV viral antigen on three occasions.[3] Twenty-one of 34 family members treated (62 percent) had a titer of 1:160 or greater, whereas only 10 to 15 percent of a random sample of healthy individuals would be expected to have anti-EBV titers of this level. In our patient anti-Epstein-Barr virus antibodies against capsular antigen (indirect immunoflorescence method) using serum obtained both during hospitalization and two weeks after discharge respectively showed high titers of 1:640 and 1:160.

The role of immunologic deficiency in patients with sinus histiocytosis has been studied in a few cases but results so far have been inconclusive. Becroft *et al.* reported a transient defect in the immunologic status of a 16-month-old boy with SHML and in his monozygous twin.[4] They also noted consistent but reversible defects in lymphocyte response to phytohemagglutinin and in augmentation of nitroblue tetrozolium reduction by neutrophils during phagocytosis. They have suggested that these defects in cellular immune functions may be important in the pathogenesis of the condition. In 1968, Becroft and Douglas reported striking histiocytic proliferation in some children with severe congenital defects in T lymphocyte mediated cellular immunity in association with thymic dysplasia.[5] Rosai and Dorfman also described thymic abnormalities in two autopsy-proved cases of SHML. It is not clear, however, if the thymus has any role in the immune deficiency state encountered in SHML.

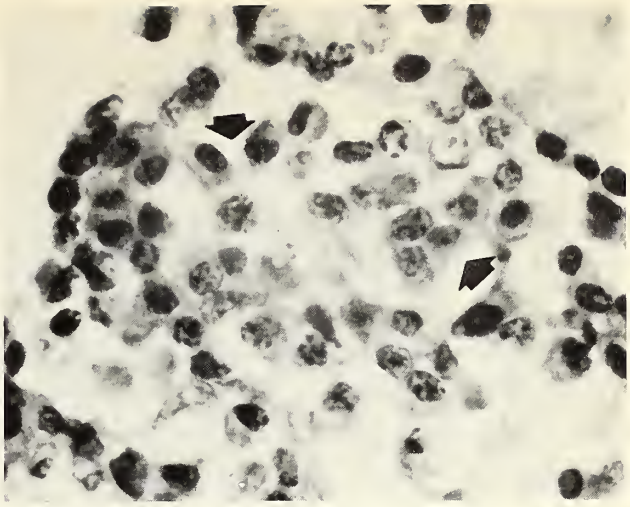


Figure 5—Compressed intersinusal tissue containing increased plasma cells. (X 840)

Immunologic function studies in our patient revealed that the whole-cell leukocyte preparation derived from peripheral blood yielded normal proportions of T and B cells. The response to phytohemagglutinin (PHA) *in vitro* was delayed. However, the magnitude of response was normal reaching maximum response at seven days. Although a delayed response is typical of lymphomatous disorders, one would also expect to find a decrease in rosette-forming cells and an increased number of B cells which was not the case in our patient.

There are two opinions concerning the cause of histiocytic proliferations. Becroft *et al.* suggested[4] that histiocytic proliferation is a result of phagocytosis of microorganisms with diminished ability to lyse them coupled with a defect in lymphocyte mediated immunity. Pickering *et al.* studied the immunologic status of two cases of SHML and found no abnormalities in humoral or cellular immune mechanism or leukocyte function.[6] However, they concluded that the possibility cannot be excluded that a breakdown in host defenses might involve a specific failure in either recognition of an antigen as foreign, or the production of one or more of the lymphocyte mediators with subsequent production of disease.

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Arteriovenous Malformations In Young Infants*

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An infant with an arteriovenous malformation (AVM) of the vein of Galen and another infant with right coronary artery to right atrial AVM developed heart failure during the neonatal period. They became asymptomatic (with digitalization) and have thrived. Spontaneous reduction in coronary arteriovenous shunt flow was documented by restudy. The natural history of these lesions suggests that spontaneous reduction in size or closure of these lesions may occur, and provides justification for deferral of surgical intervention in selected cases.

Congential cardiovascular malformations have become increasingly amenable to surgical correction in early infancy. A rational choice between medical and surgical therapy is based upon assessment of a lesion's threat to life in a given patient, as well as on knowledge of its natural history.[1]

Congenital arteriovenous malformations (AVM) large enough to cause heart failure in the neonate are sufficiently rare as to preclude a systematic study of their natural course. Early death has been reported in infants with large AVM, so that surgical correction is commonly attempted when technically feasible, with mixed success depending upon the location and size of the lesion.[2-9]

Published experience, however, suggests that arteriovenous fistulas, diagnosed in older children or adults, may undergo spontaneous reduction in size, or closure.[10-14] It is the purpose of this report to present two cases in which neonatal heart failure due to congenital arteriovenous fistula responded promptly to medical therapy and gave no evidence of local or cardiovascular progression through infancy.

CASE 1—CEREBRAL AVM

This male infant weighed 3100 grams and was apparently healthy at birth. A cardiac murmur was heard at one month of age. At ten weeks of age he developed congestive heart failure. A grade 2/6 blowing systolic murmur was present along the left sternal border. The chest roentgenogram showed cardiac enlargement and normal pulmonary vascular markings. The electrocardiogram showed right ventricular enlargement. Digitalization resulted in improvement. On the third hospital day a continuous cranial bruit was detected. Cardiac catheterization revealed arterialization of blood draining into the superior vena cava (Table 1). A large arteriovenous malformation of the vein of Galen was demonstrated angiographically (Figure 1).

The infant was maintained on digoxin and has grown and developed normally. The head circumference has remained at the 90th percentile, while height and weight are at the 50th percentile at age 2-1/2 years. There has been no evidence of neurological abnormality. Computerized tomography of the head at 2-1/2 years showed a circular midline mass lesion measuring 3.2 cm in diameter, having the density of blood, and located just posterior to the third ventricle (Figure 2).

CASE 2—CORONARY AVM

This male infant was born at term after an uncomplicated pregnancy and delivery. He weighed 3.2 kg at birth. On the second day of life tachypnea and a blowing, continuous, grade 3 shunt murmur at the right sternal border were noted.

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Table 1
Cardioc Cotheterizotion Doto, Potient #1

Location	Hb O_2 Saturation (%)	Pressure (mm Hg)
Internal jugular vein	85*	
Left innominate vein	75	
Superior vena cava	84*	
Right atrium	79	mean = 3
Right ventricle	78	28/3
Pulmonary artery	78	28/6; mean = 15
Left ventricle	96	115/8
Femoral artery	96	115/60; mean = 85

*High saturation values in jugular vein and superior vena cava represent shunt of arterial blood through the arteriovenous fistula at the vein of Galen. Remainder of catheterization data is within normal range.

Table 2
Cordioc Cotheterization Doto, Patient #2

	Age 4 days		Age 18 mos	
Location	Hb O_2 Sat (%)	Pressure (mm Hg)	Hb O_2 Sat (%)	Pressure (mm Hg)
Superior vena cava	54		67	
Right atrium	75*	m = 3	75*	m = 5
Right ventricle	77	80/15**	75	25/3
Pulmonary artery	80	70/40; m = 52**	73	22/8; m = 15
Pulmonary vein	95			
Left atrium	93	m = 4		
Left ventricle	95	68/18		
Aorta	91	70/40; m = 53	94	100/52; m = 75
Qp : Qs	2.4		1.3	
Rp : Rs	0.4		0.1	

*Increment in saturation at right atrial level reflects shunt of aortic blood through the right coronary artery and fistula into the right atrium.

**High right ventricular and pulmonary arterial pressure reflect persistent pulmonary hypertension due to high pulmonary blood flow in the neonatal period. Normal values at this age are 25-35/0-10 mm Hg in right ventricle, and 25-35/12-20 in pulmonary artery. The pulmonary to systemic flow ratio (Qp:Qs) is normally 1.0, and the resistance ratio (Rp:Rs) is normally 0.2.

In addition, the signs of congestive heart failure were present. The chest roentgenogram (Figure 3) showed marked cardiomegaly and increased pulmonary vascularity. The electrocardiogram demonstrated right axis deviation, right atrial, and right ventricular enlargement. The infant improved after digitalization. Cardiac catheterization on the fourth day of life (Table 2) demonstrated aneurysmal dilatation of the right coronary artery with a fistulous tract draining into the right atrium (Figure 4). A small patent ductus arteriosus was also present.

Moderate tachypnea persisted, but the infant was able to feed and gain satisfactorily. Surgical intervention was considered, but a trial of medical management was elected. Digoxin maintenance has been continued and at age 1-1/2 years the infant continues to thrive and is free of signs of heart failure. Cardiac catheterization was repeated at age 18 months. The ductus arteriosus was closed, and the coronary arteriovenous fistula was angiographically unchanged. The pulmonary/systemic flow ratio was almost 50 percent smaller than initially. Hemodynamic measurements demonstrated a fall in right ventricular and pulmonary arterial pressure from systemic level to normal (Table 2).

DISCUSSION

An arteriovenous fistula provides a low-resistance diversion of blood flow resulting in high-output circulatory congestion and progressive dilatation of vessels feeding and draining the lesion, together with a stimulus to the development of collateral channels. Cerebral AVM may be so large that intractable congestive heart failure occurs in the neonate. Such cases usually are characterized by pulmonary hypertension at systemic level, due to a combination of large pulmonary blood flow and neonatal elevation of pulmonary vascular resistance.[3] When cardiac failure develops beyond the neonatal period, hemodynamic measurements show a normal or mild elevation of the pulmonary arterial pressure.[2,3] Unlike central left-to-right shunts, such as septal defects or patent ductus arteriosus, peripheral fistulous connections are independent of maturational changes in pulmonary vascular resistance. Therefore a delayed onset of heart failure, as in our case of cerebral AVM, probably reflects a smaller fistula initially, and possibly a progressive increase in shunting due to enlargement of or recruitment of additional feeding vessels.

The rarity of neonatal cardiac failure in coronary AVM may be attributed to relative infrequency of the lesion and to the presence of a single arterial feeder. In addition, coronary AVM presents a central shunt draining directly into the right heart. As such the quantity of flow through the fistula may be restricted by neonatal elevation of right heart outflow impedance.

Instances of spontaneous closure or near-closure of both cerebral and coronary AVM's in older individuals suggest that forces within the malformation may be operative tending progressively to reduce shunt flow.[10-14] Thus, while hemodynamic factors tend to enlarge the lesion and increase



Figure 1—Case 1—Cephalic phase of aortogram showing aneurysmally dilated vein of Galen(A), dilated venous sinuses(V) (left panel, frontal view), and arterial supply(a) (right panel, lateral view).

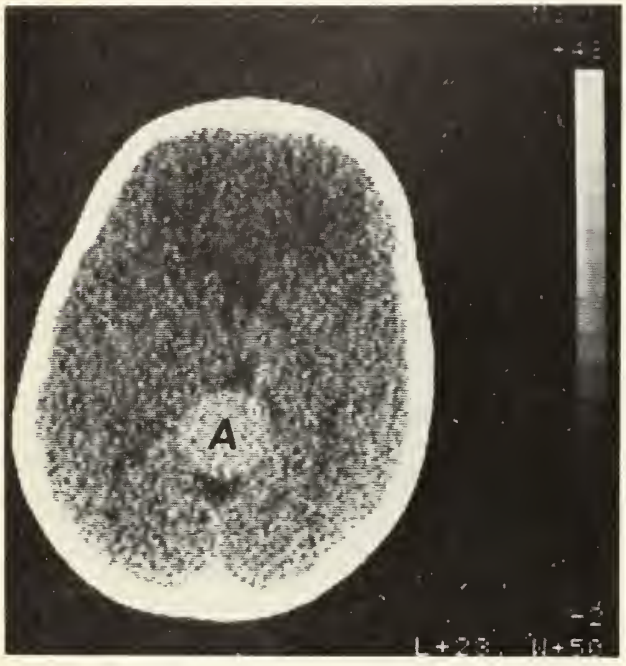


Figure 2—Case 1—Computerized tomographic scan of head at age 18 months showing circular midline mass(A), 32 mm diameter, just posterior to third ventricle, representing aneurysmally dilated vein of Galen. There is no evidence of hydrocephalus.

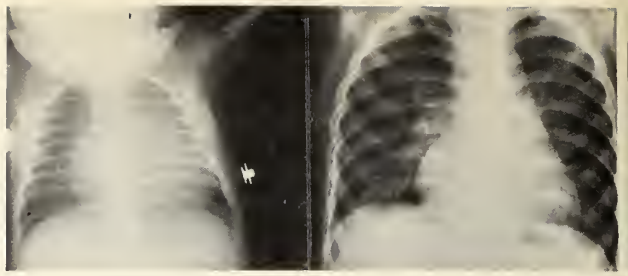


Figure 3—Case 2—Chest roentgenogram at age 3 days (left panel) showing cardiomegaly, and decrease in heart size at age 18 months (right panel).

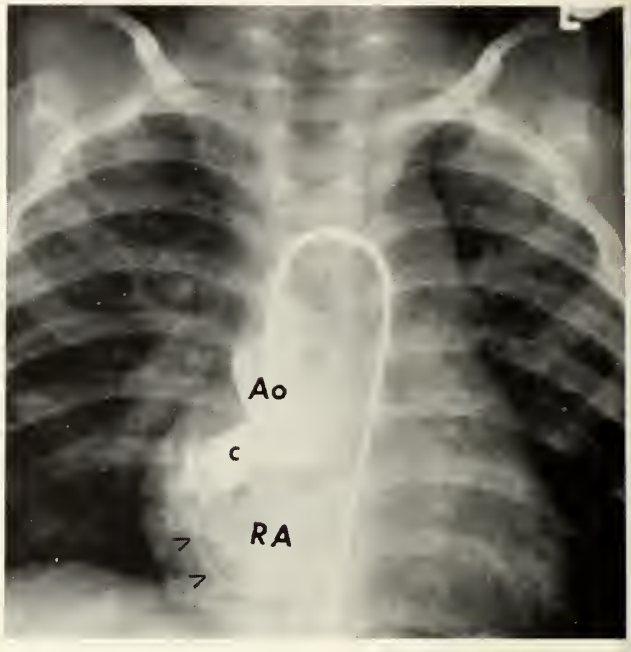


Figure 4—Case 2—Aortogram(Ao) showing aneurysmally dilated right coronary artery(c), opacification of right atrium(RA), and right coronary artery continuation (arrow heads) inferiorly and medially toward apex of heart.

the shunt, flow velocity and turbulence within the fistula can cause proliferative changes leading to occlusion. Such alterations have been observed at sites of vascular narrowings and are proposed as an explanation for the localization of intra-arterial atheromatous changes at branching sites.[16,17]

The choice of medical versus surgical management of AVM is governed by hemodynamic considerations and by local effects of the lesion. This is particularly important in cerebral AVM, in which deformation of neighboring structures may lead to a variety of neurological complications. The most common of these is hydrocephalus due to mechanical obstruction of the aqueduct of Sylvius by the dilated vein of Galen. In such instances surgical relief is essential, but it either may relieve or further complicate the patient's cardiovascular status.[5]

There is insufficient experience on which to base an assessment of the surgical indications in a young infant with a large coronary AVM. From a technical standpoint, the capability of eliminating the fistulous run-off with minimal risk is available, at least in older individuals. This accomplished, however, the subsequent history of the aneurysmally dilated right coronary artery as in our case #2, is conjectural. It is likely that dilatation of this vessel is acquired in response to enormously increased blood flow.[18] The catheterization data obtained on the patient in case #2 at four days of age suggest that shunt flow through the right coronary artery, which normally carries only two to four percent of the left ventricular output[19] was at least equal to total aortic flow. Reduction in shunt flow, whether spontaneous or accomplished surgically, at least should eliminate this stimulus to further dilatation of the artery. In case #2, the deferral of surgical intervention was based on (1) the prompt control of heart failure by digitalization, (2) the absence of evidence of coronary insufficiency, (3) evidence on repeat catheterization 18 months later of substantial spontaneous reduction in shunt flow, and (4) a recent report of spontaneous near closure of a coronary AVM in a 14-year-old patient.[10]

SUMMARY AND CONCLUSION

Presented are two cases of heart failure in early infancy due to arteriovenous malformation, one involving the vein of

Galen and the second a right coronary artery to right atrial fistula. Both infants responded promptly to digitalization. Their subsequent course, during 2-1/2 years and 1-1/2 years, respectively, demonstrated absence of local or cardiovascular complications, normal growth and development, and spontaneous reduction in coronary fistula flow. In the light of other reports of spontaneous reduction in size or closure of such lesions we continue to follow these patients medically. These reports provide natural history data concerning these unusual lesions which may prove useful in assessing the indications for surgical intervention in other similar cases.

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REVIEW ARTICLE

Surgical Treatment of Glaucoma

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A classification of the glaucomas and a description of the present surgical treatment procedures are reviewed.

A review of the classification of the glaucomas is necessary in order to understand both medical and surgical approaches to treatment. The glaucomas are classified into: (1) angle closure—acute, subacute and chronic; (2) open angle; (3) secondary; and (4) congenital.

Angle closure glaucoma, which accounts for approximately 10 percent of the glaucomas, may be acute, with the typical manifestations of blurred vision, severe pain, red eye, hazy cornea, and dilated pupil. It may present with episodes of slight headache, blurred vision, and possibly a dilated pupil; or it may be a chronic condition with moderately elevated pressures and periods of exacerbation and remission. In the latter, patients often are aware of increased pressure because of slight pain and haloes around lights.

In the *acute angle closure type,* the definitive therapy is surgical. Medical treatment with carbonic anhydrase inhibitors, miotics, osmotic agents, and local steroids is employed first before an iridectomy or a filtering procedure is performed. Some patients can be treated medically; however, the attack rate on a five-year follow-up is 50 percent. Because of this, many practitioners advocate a prophylactic surgical procedure on the uninvolved eye. The more chronic cases of narrow angle glaucoma usually require a filtering operation if medical treatment fails to control the intraocular pressure.

In *chronic open angle glaucoma* most ophthalmologists depend primarily on medical treatment. The diagnosis depends on a triad of elevated intraocular pressure, typical visual field changes and glaucomatous cupping of the optic disc (Figures 1 and 2). Surgery is performed only if maximum medical therapy fails to control the above factors or if noncompliance is a problem. Medical therapy may consist of miotics, sympathomimetic drugs and carbonic anhydrase inhibitors. Newer eyedrops, such as beta-blocking agents and the cannabis alkaloids give promise of significant aid to medical therapy. However, if surgery is indicated, some form of filtering procedure is performed.

Perhaps the most difficult of the glaucomas to manage, both medically and surgically, are the large group of cases where other ocular conditions are complicated by elevated intraocular pressure. Secondary glaucoma may follow trauma, other surgical procedures, inflammation of the uveal tract, swelling or leaking cataracts, neovascular conditions following vascular occlusions or diabetic retinopathy, tumors, and congenital defects.

Obviously, treatment of the initiating condition is undertaken, and only when this fails to protect the eye is glaucoma surgery indicated. The type of surgery is dependent on the cause and anatomical conditions present. Following trauma, careful surgical repair may prevent complications leading to secondary glaucoma. Hemolysis leading to glaucoma may be cleared with irrigation-aspiration techniques. In some in-

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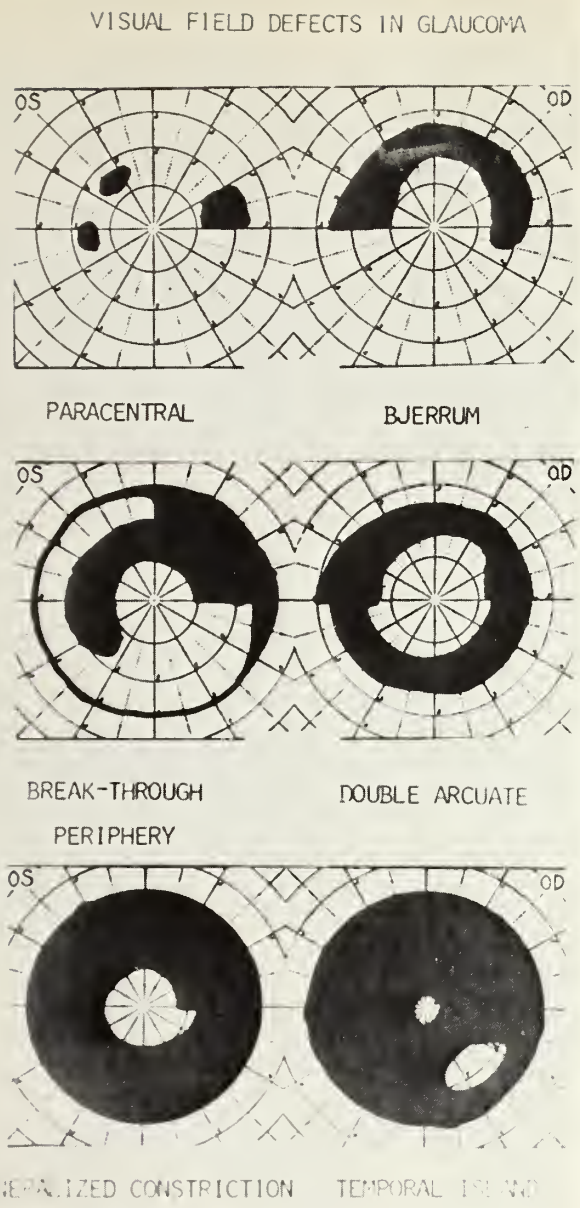


Figure 1—Progression of visual field defects.

stances iridectomies can correct a pupillary block; in others, a filtering operation is necessary, and in some the lens has to be removed. In neovascular glaucoma destruction of the ciliary body with a laser, cryoprobe or diathermy needle might reduce the pressure. Laser treatment of the new vessels on the iris and retina offers new hope in this common and difficult problem.

Congenital glaucoma may present early in life with large corneae (greater than 12 mm in horizontal diameter), cracks in Descemet's membrane, a sometimes hazy cornea, and photophobia. If the cornea is clear, a goniotomy usually is performed. Using a contact lens and a microscope a small knife is introduced across the anterior chamber to the easily visualized angle, where a 120° incision into Barkan's membrane, which covers the trabeculum, can be made. This can be repeated if the pressure is not controlled. In goniotomy failures of patients with corneal scars, a trabeculectomy or other filtration operations can be performed.

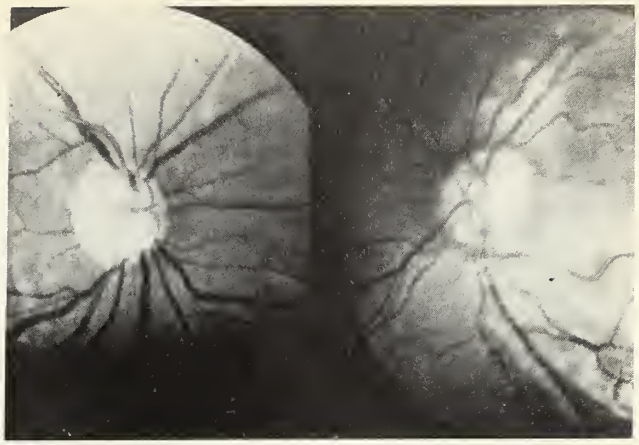


Figure 2—The right optic disc shows marked cupping; the left disc has only moderate cupping.

PROCEDURES

Surgical iridectomies are performed in order to permit aqueous drainage from the posterior to the anterior chamber and thus open the angle. This operation generally is performed through a small incision at the corneo-scleral junction. A newer method of creating an opening in the iris is with the laser beam. Drawbacks in this procedure are that in five to ten percent of the cases the laser fails to penetrate the iris, the iridotomy opening sometimes closes, and occasionally an acute attack can be provoked. With the development of newer lasers, this ultimately may become the procedure of choice since it does not require a surgical incision into the eye.

Several filtering operations have been developed over the years. These procedures include trephination, iridencleisis, and scleral cautery, all of which create a drainage channel from the anterior chamber to the subconjunctival space. They generally are effective in approximately 85 percent of the cases. However, the major deficiencies of these procedures are the development of large cystic filtering blebs, which are potentially hazardous, and the formation of cataracts in approximately 40 percent.

A newer procedure which has been popularized in the past ten years, because of refinements in microsurgical techniques, is the trabeculectomy or subscleral sclerectomy (Figure 3). In this operation a lamellar scleral flap is made under the conjunctiva into the cornea. A section of the anterior chamber angle is then excised under this flap. This permits aqueous to drain diffusely along the created scleral bed and then under the conjunctiva. Thus a more physiological drainage system is created. The major advantages of this procedure are a diffusely elevated filtering bleb and a lower incidence of cataract-forming flat anterior chambers. Some investigators have suggested that this procedure is more effective in black patients, who are also more difficult to control medically.

SUMMARY

The type of glaucoma surgery to be performed should be custom-tailored for the problem at hand. Research through the years has elucidated the mechanism of some glaucomas and has afforded rational surgical approaches in their treatment. We hope future developments will reveal the exact location and nature of the blockage in open angle glaucoma and suggest appropriate surgical measures for its treatment.

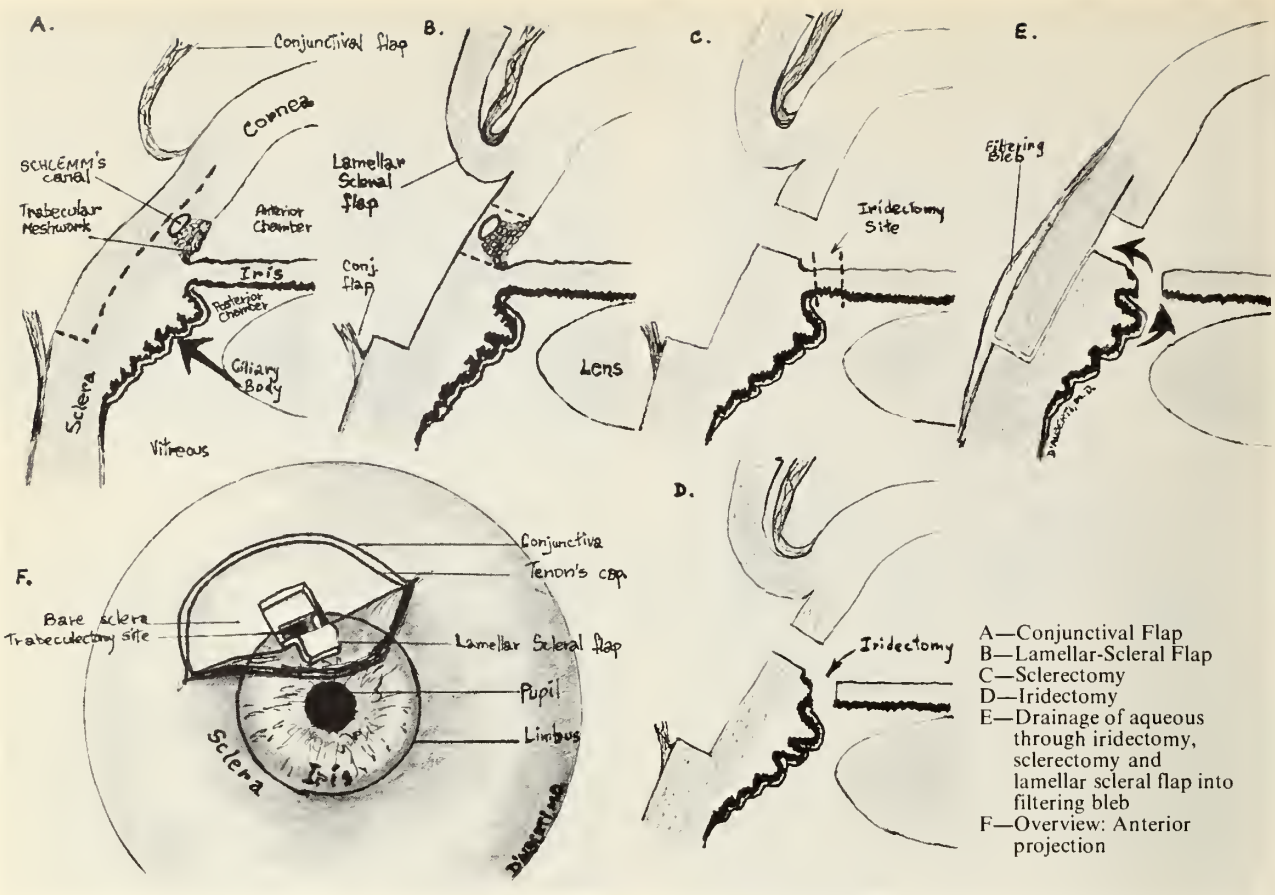


Figure 3—Trabeculectomy procedure.

Acknowledgment is given to Claude F. D'Alberti, M.D., for the drawing of the trabeculectomy procedure.

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The Feingold Hypothesis: Current Studies

J. PRESTON HARLEY, Ph.D.
CHARLES G. MATTHEWS, Ph.D., Madison, Wisconsin

In recent years, a great deal of clinical-research interest and effort has been directed to the study of hyperactive children.[1-3] These children usually are described as being significantly more restless, easily distracted, inattentive, and emotionally labile relative to their peers. The hyperactive child is most likely to become an object of attention in situations which demand sedentary/attentive behavior. As a consequence, the majority of these children first come under medical-psychological evaluative scrutiny because of academic difficulties and behavior problems at school.

The reported incidence figures for hyperactivity vary widely depending upon the various diagnostic criteria that have been employed. Estimates have been offered ranging from 5 to as high as 20 percent of school age children as being hyperactive. Hyperactivity is much more common among males than females. Suspected or presumed etiology includes perinatal, psychological, environmental, neurotransmitter and genetic factors, singly or in combinations. The major treatment has been the use of stimulant medication and, more recently, behavior modification programs. Other treatments which have been tried with mixed success and based upon limited scientific support include: megavitamins, caffeine, elimination of fluorescent lighting in the classroom, and amelioration of allergies. A current and well-publicized treatment, which is the subject of the present article, is the use of diet to modify hyperactivity in children.

THE FEINGOLD HYPOTHESIS

Dr. B. F. Feingold of the Kaiser Permanente Medical Center in San Francisco has postulated that approximately 30 to 50 percent of hyperactive children demonstrate significant improvement in behavior, often quite dramatically, when placed on a special elimination diet program[4-5]. He recommends removing foods containing synthetic food colors and flavors and naturally occurring salicylates. After placing their child on this diet program, parents frequently have reported impressive improvements in their child's previously hyperactive behaviors. Once maintained on the diet, even minute quantities of prohibited foods are reported to cause a return of the disruptive behaviors within several hours and with the undesirable behavior lasting up to several days even following a single dietary infraction. This observation has prompted Dr. Feingold to state that he is capable of turning undesirable behaviors such as juvenile delinquency "on and off" at will. In addition to being beneficial to the hyperactive child, Dr. Feingold has claimed success in the dietary treatment of other disruptive behavioral patterns associated with learning disabilities, epilepsy, and mental retardation.[6] Furthermore, a causal relationship has been implied between the increasing amounts of additives in the food supply over the last several decades and the increasing number of reports of hyperactivity. Finally, the use of a labeling logo has been suggested by Dr. Feingold to help the consumer readily identify food products which do not contain artificial colors or flavors[5].

These very provocative claims and recommendations have been derived primarily from uncontrolled clinical observations and anecdotal reports. If verified, the Feingold hypothesis obviously would have widespread implications not only for clinical management of a variety of undesirable behaviors but for the food industry as well. Therefore, two independent groups of food, medical, and behavioral scientists were formed to conduct comprehensive reviews of the evidence relevant to the Feingold hypothesis[7,8]. The general conclusions offered by these committees included the observation that controlled research was lacking, which would be a requisite for any definite statements to be made regarding the efficacy of the diet in ameliorating hyperactive symptoms.

Conners and his colleagues[9] completed the first experimental investigation of the Feingold hypothesis. Their results indicated that a significant improvement in subjective teacher ratings was found in 6 to 15 subjects who were given the control diet for 4 weeks followed by 4 weeks on the Feingold diet; but the effect was not evident in those individuals receiving the reverse diet sequence. A critique[10] of the study by the National Institute of Education, who had funded Conner's investigation, warned that the results of the experiment should be cautiously interpreted for several methodological reasons.

An investigation at the University of Wisconsin studied 36 hyperactive boys, age 6 to 12, and 10 hyperactive boys, age 3 to 5. Food for the entire family was provided for the duration of the study. Half of the children (and their families) first were placed on a diet consisting of only those foods permitted in Dr. Feingold's book and the other children were given menus which included ordinary amounts of artificial

*Reprinted with permission of *Contemporary Nutrition* 3:4 (April) 1978, a newsletter from the Nutrition Department of General Mills, Inc., Minneapolis. Drs. Harley and Matthews are on the staff of the Neuropsychology Laboratory, Department of Neurology, University of Wisconsin Center for Health Sciences, Madison, Wisconsin.

colors and flavors. After four weeks, the children were switched over to the other diet. The diets were comparable in appearance, taste, and palatability. Neither the children, their families, nor any members of the investigative team knew which diet a particular child was receiving at any point in the experiment. To assure objectivity, the children were assigned to their diet sequence by using a code which remained secret until the study was completed. Prior to beginning the diet phase of the investigation, each child was given neurological and psychological examinations and hyperactivity ratings were obtained from parents and from the child's classroom teacher. Classroom observers carefully recorded the frequency of restless, inattentive, disruptive, and aggressive behavior displayed by the hyperactive child and made similar observations upon a "control" non-hyperactive classmate selected by the classroom teacher. The children's activity level also was measured in a standardized laboratory setting. The psychological tests, which included measures of attention, concentration, reaction time, and motor coordination, and the laboratory observations were repeated at the end of each diet period. Parent and teacher ratings on degree of hyperactivity were made each week and observations were made in the classroom twice per week throughout the study.

The overall results of this study did not provide convincing support for the Feingold diet, certainly not in the larger group of 36 boys, 6 to 12 years of age. The frequency with which favorable diet effects were judged to be present was highest in the subjective parent ratings, declined sharply in the teacher ratings and essentially disappeared in the objective psychological, classroom and observational data. It was also noted that the parental reports of improved behavior on the Feingold diet occurred only when the child was on the ordinary diet first, followed by the Feingold diet but not when the Feingold diet was given first and then followed by the controlled diet, the same diet order effect that was reported by Conners *et al.*[9]

The smaller group of ten preschool-age boys showed a stronger tendency to receive parental ratings which were in the direction of improved behavior when on the Feingold diet, even though the parent ratings were not supported by improvement in psychological tests or in the laboratory grid room activity measurements. However, more detailed study of a larger group of preschool children was recommended by the Wisconsin team.

A follow-up experiment was conducted utilizing the research protocol recommended by the Interagency Collaborative Group on Hyperkinesis.[8] In the second study, nine children were selected from the group of 46 boys used in the first experiment. The same kinds of parent, teacher, classroom observational and psychological data obtained in the first study were gathered during a two-week period while the children and their families remained on their ordinary diet and again following two weeks on the Feingold diet. As in the first study, all the food required for the entire family was provided for the duration of the project. The nine families were then continued on the Feingold diet for 9 weeks, during which time five of the children daily ate specially prepared cookies and candy bars containing a blend of all the certified food colors in an amount equivalent to the average daily intake. The other four children ate treats of similar taste and appearance but which contained no artificial colors. For a nine-week period, the children were daily given either color-containing or color-free treats. One type of treat was given for two weeks and then the other treat was administered for two weeks. The last treatment condition was three weeks in duration. As in the first study, none of the parents, teachers, classroom observers or research team staff members knew which child was receiving which kind of treat at a particular time during the experiment. Based upon the clinical observations reported by Dr. Feingold that hyperactive behaviors are exacerbated by foods containing artificial colors, it was predicted that the children would show a significant worsening of behavior during the challenge as compared to the placebo periods.

The results of the follow-up study again provided little support for Dr. Feingold's assertions. One of the nine children seemed to react adversely to the color-containing food but the other eight did not; in fact, parents and teachers frequently rated the children as more hyperactive during the periods in which the color-free treat was being given. Analysis of the psychological test measures of attention and motor coordination, and analysis of the classroom observational data obtained twice per week under the color versus color-free conditions gave no evidence of differences in performances or behavior in either treatment condition.

The overall conclusion of the two Wisconsin studies was that the investigations had been unable to detect the kind, frequency, or degree of changes in behavior related to artificial food colors described by Dr. Feingold. While the possibility that a small group of children may be affected adversely by food additives certainly deserves further study, the hypothesis that hyperactivity in children is frequently or generally the result of artificial food colors and flavors was not supported by the results of objectively conducted experiments using diets based on Dr. Feingold's guidelines.

Preliminary reports of similar challenge experiments conducted by Drs. Conners and Goyette at the University of Pittsburgh also showed that the challenge food materials did not substantially affect the general symptoms of the children although a brief and transient decrement in performance on a measure of attention was found.[12] The Pittsburgh investigators noted that younger children may be more sensitive to food substances than are older children.

The experimental data accumulated to date (currently additional studies are being conducted in California and in Australia) have not provided reasonable scientific support for stating that a significant relationship exists between food additives and hyperkinesis. An expert committee on hyperkinesis and diet in a report[13] to the Nutrition Foundation concluded that there is no urgent need for a change in food manufacturing or labeling practices based upon the presently available data. A moratorium on additional public advocacy has been called for by others[14] until the completion of appropriate supportive data derived from rigorously designed, carefully controlled studies.

DISCREPANCY BETWEEN CONTROL STUDIES AND CLINICAL REPORTS: IILLUSION, ARTIFACT OR REALITY?

The Feingold diet clearly offers an appealing, relatively simple treatment for a very large number of children who have rightly or wrongly been labeled hyperactive. Parents are typically attracted to the diet treatment program because of dissatisfaction with other therapies that have been tried. They are frequently seeking alternatives to medication and other treatment modalities. The numerous Feingold associations, which have been established across the country, also contribute to a positive expectancy effect. The inclusion of any new diet can cause direct placebo effects as well as subtle changes in the total family situation.

Nonetheless, it must be emphasized that when data are collected via rigorous experimental designs which minimize potential sources of subjective bias and positive behavioral outcome expectancy, diet-related behavioral changes are much less spectacular than are the impressive clinical case studies or fervent parental testimonials which have been so widely disseminated.

By its very nature, experimental research cannot offer absolute answers to the issue being investigated here; nonetheless, it is now evident that numerous attempts by scientists in the United States, Canada, and Australia to document the spectacular claims that have been associated with dietary intervention have been in large part unsuccessful. While little research support has been found for these clinical reports with respect to school age children, some data do suggest that there may be a small subgroup of younger children who display adverse behaviorial reactions to particular food substances, and hyperactive children in this age group clearly deserve further study. Firm scientific conclusions regarding this entire investigative area have not yet been reached and may not be for a considerable period of time. A consensus has emerged, however, among reputable investigators that advocating sweeping treatment recommendations or mandating changes in national food manufacturing and distribution practices is distinctly premature at this time.

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THERAPEUTIC DRUG INFORMATION

This information is compiled by the Schwartz Inter-National Pharmaceutic and Therapeutic Drug Information Center of the Brooklyn College of Pharmacy, Long Island University.*

1. Please provide information concerning the use of doxycycline in the treatment of travelers' diarrhea.

Travelers' diarrhea is a world-wide illness caused by a variety of pathogens, the most common of which is enterotoxigenic *Escherichia coli.*[1] It is most prevalent in tropical and sub-tropical areas especially in Mexico, North Africa, and the Middle East where it seems to be related to poor sanitation and hygiene.[2] The condition in most individuals is short lived, usually lasting one to three days. It represents itself with a sudden onset of loose stools with variable symptoms including nausea, vomiting, and abdominal cramps. In severe cases, however, it may be necessary to hospitalize the patient.

Symptoms of travelers' diarrhea may be reduced by the combination of diphenoxylate and atropine (Lomotil)® or other opiates. However, these drugs may aggravate diarrhea whose etiological cause is Shigella or Salmonella organisms. Antibiotics such as neomycin (Mycifradin)® and sulfonamides attempted for prophylactic use in travelers' diarrhea have been reported to decrease attack rates.[3] Tetracycline (Achromycin V,® Panmycin,® Sumycin)® has been used extensively as a broad-spectrum antibiotic for oral treatment of many common gastrointestinal infections including cholera.

Sack *et al*[4] have reported a double-blind study of the efficacy of doxycycline (Vibramycin)® in preventing travelers' diarrhea. Doxycycline is one of several antibiotics belonging to the family of tetracyclines. It has the advantage of excellent oral absorption and a prolonged half-life that permits once-a-day dosing. Doxycycline also has been shown to be effective in certain *Escherichia coli* isolates resistant to other tetracyclines.[4] In this study, 39 Peace Corps volunteers received doxycycline or placebo during their first five weeks in Kenya. The doxycycline group received 200 mg while in flight to their destination and 100 mg daily during the first three weeks. The control group received a placebo in a similar fashion. Nine of the 21 volunteers taking placebo and only one of the 18 volunteers taking doxycycline developed travelers' diarrhea during the period of treatment. The protection persisted for at least one week after the doxycycline treatment was discontinued.

In conclusion, doxycycline appears to be highly effective in preventing travelers' diarrhea associated with enterotoxigenic *Escherichia coli,* the most common pathogen implicated in this condition. Since different enteric pathogens can be responsible for travelers' diarrhea in various geographic areas of the world, the precaution of consuming food that has been properly stored and cooked, and water that has been properly treated is highly recommended.

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2. Is there a clinically significant drug interaction between chlorpromazine and lithium carbonate?

Chlorpromazine (Thorazine)® and lithium carbonate (Eskalith,® Lithane,® Pfilithium®) are utilized concurrently in certain clinical situations on both a short and long-term basis.[1] There have been reports involving studies in laboratory animals and man that such a combination, when both drugs were administered orally, resulted in lower chlorpromazine levels than normally would be expected.

Rivera-Calimin[2] studied the lithium-chlorpromazine interaction in rats and noted that the combination resulted in lower plasma and brain levels of chlorpromazine than animals that received chlorpromazine alone. In addition, the amount of chlorpromazine that remained in the stomach of animals who received the combination was significantly higher than animals that received chlorpromazine alone. Gastric emptying time was also shown to be inhibited by lithium.

*The Center serves as a source of intelligence on therapeutic and pharmaceutic information not readily available to physicians, at no charge to them, and provides this information with minimal time involvement. It is staffed by trained pharmacists; Jack M. Rosenberg, Pharm.D., Associate Professor and Chairman, Division of Clinical Pharmacy, Brooklyn College of Pharmacy, is Director and Walter Modell, M.D., Emeritus Professor of Pharmacology at Cornell University Medical College, is pharmacologist consultant. The service is available Monday through Friday from 9 a.m. to 4:30 p.m.—telephone (212) 622-8989 or 303-2735. Responses to these questions were prepared by J. M. Rosenberg, M.S.; Pharm.D.; D. Sherman, M.S., P. Sangkachand, M.S., R.Ph.; Doris Lau, B.S.

These same authors have reported their observations of lower chlorpromazine levels in patients on concomitant lithium therapy.[3] Patients who ingested 400 to 1000 mg of chlorpromazine which ordinarily produces plasma levels between 100 to 300 ng/ml or more had plasma levels of 58 to 70 ng/ml.

A study of the lithium-chlorpromazine interaction on seven healthy males was conducted in a randomized crossover study.[4] Each subject ingested two doses of chlorpromazine (100 mg) as the liquid concentrate: one prior to lithium therapy, and one after a seven-day treatment with lithium (900 mg/day in divided doses). After treatment with lithium, mean plasma levels of chlorpromazine were lower at all times than the corresponding plasma levels of chlorpromazine prior to lithium therapy. Peak chlorpromazine levels were about 40 percent lower after lithium treatment, but there was no difference in time to reach peak plasma levels. The area under the chlorpromazine concentration-time curve was 26.6 percent smaller. The authors hypothesize that impaired gastric emptying resulted from lithium and may account for the diminished chlorpromazine levels; however, a different study of lithium's effect on gastric emptying in man did not consistently support this theory.[5]

Although the precise mechanism of interaction between chlorpromazine and lithium is not known, there is some evidence that the combination resulted in lower chlorpromazine levels than normally would occur. The significance of this interaction is that the chlorpromazine response may be less than expected during combined therapy and/or chlorpromazine toxicity could arise upon withdrawal of lithium from a patient receiving this combination.

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3. Do you have any information concerning the use of levamisole in the treatment of cancer?

Levamisole, an effective anthelmintic with few toxic effects, is available as a veterinary product (Tramisol®) in the United States from American Cyanamid. This orally active agent has attracted attention because it was found to possess immunotropic properties. It seems to restore inefficient host defense mechanisms and thus its usefulness as an adjunct in the treatment of certain types of human cancers as well as other conditions is being explored.

In most animal cancer models, levamisole does not influence the growth of primary tumor but prolongs the remission period after chemotherapy. *In vitro,* levamisole was found to restore azathioprine (Imuran®) suppressed T-cell function. In patients with cancer, levamisole restores skin reactivity to 2, 4 dinitrochlorobenzene (DNCB) and purified protein derivative (PPD). In those with Hodgkin's disease, levamisole restores the ability to form E rosettes.[1,2] The above are all indications of enhancement of cell-mediated immunocompetency.

A long-term, multicenter, double-blind study in patients undergoing operations for primary bronchial carcinoma was designed to test the immunotropic effects of levamisole.[2] The patients received levamisole or placebo for three days every two weeks, starting three days before surgery. Unless there was clinical evidence of recurrence, cytostatic drugs, corticosteroids, and radiotherapy were prohibited. In the 111 patients who had been followed for one year, the incidence of side effects was similar in both the placebo and drug treatment groups. Recurrences occurred in ten out of 51 patients (seven deaths) that received levamisole and in 20 out 60 (twelve deaths) that received placebo. The difference appears not to be due to chance alone. In the levamisole group, there were fewer recurrences in patients with squamous cell carcinomas and medium and large primary tumors, and fewer suspected and proved recurrences and deaths from metastases in patients with extended tumors. Distant recurrences (in bone, brain, and liver) tended to be less common in the levamisole group, whereas the disease-free interval (time between surgery and evidence of recurrence) in relapsing patients was almost identical in the two groups.

Rojas *et al*[3] conducted a clinical trial of levamisole in women with primary inoperable breast cancer (stage III). After being rendered clinically disease free by radiotherapy to the breast, supraclavicular area, and axilla, patients were allocated alternately to a control group (no further treatment) or to a levamisole-treated group. The follow-up in 43 patients (20 levamisole treated and 23 controls) indicated significant prolongation of the median disease-free interval (25 vs. 9 months) and survival (90 vs. 35 percent alive at least 30 months) in the levamisole-treated group compared with the controls. Levamisole treatment also was associated with an increase in the percentage and intensity of delayed hypersensitivity skin reactions and in the absolute lymphocyte counts. Although no significant toxicity of levamisole was observed, a slightly higher incidence of nervousness, gastric intolerance, and fever has been reported.

In conclusion, levamisole seems to stimulate the cell-mediated immune response and thus may be a valuable adjunct in the treatment of certain types of human cancers.

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(See housing application—p. 144, this issue)

DAILY SCHEDULE

Friday, May 11, 1979
3:30 p.m. —Board of Trustees

Saturday, May 12, 1979
8:00 a.m. —Registration Opens
9:00 a.m. —Message Center Opens
11:30 a.m. —Meeting—NJ Committee on Trauma
11:30 a.m. —Luncheon—NJ Orthopaedic Society
12 noon —Scientific and Informational Exhibits Open
12 noon —Luncheon—NJ Committee on Trauma
1:00 p.m. —Annual Spencer T. Snedecor Trauma Oration
2:00 p.m. —Motion Picture Theatre
2:00 p.m. —House of Delegates
3:30 p.m. —Reference Committees: Constitution and Bylaws, "A", "B", "D", "G"
6:00 p.m. —Reception for Alumni—Jefferson Medical College

Sunday, May 13, 1979
7:15 a.m. —Annual Prayer Breakfast
8:00 a.m. —Registration Opens
8:00 a.m. —Scientific Session on Oncology
9:00 a.m. —Message Center Opens
9:00 a.m. —Scientific and Informational Exhibits Open
9:00 a.m. —Scientific Sessions:
Allergy, Emergency Medicine, Medicine
Dermatology, Obstetrics and Gynecology
Family Practice, Pediatrics
Neurosurgery and Neurology
Psychiatry
9:30 a.m. —Meeting—NJ Academy of Ophthalmology and Otolaryngology
10:00 a.m. —Motion Picture Theatre
10:00 a.m. —Reference Committees: "C", "E", "F", "H"
11:30 a.m. —Luncheon—NJ Academy of Ophthalmology and Otolaryngology
12 noon—Luncheon—NJ State Urologic Society
12:15 p.m. —Luncheon—NJ Chapter, American College of Emergency Medicine
1:00 p.m. —Luncheon—Oncology Society of NJ
1:00 p.m. —Scientific Sessions:
Anesthesiology
Cardiovascular Diseases, Medicine, Nuclear Medicine
Ophthalmology
Otolaryngology
Plastic and Reconstructive Surgery
Urology
2:00 p.m. —Motion Picture Theatre
3:00 p.m. —House of Delegates
4:30 p.m. —Meeting—Widows and Orphans Society
6:30 p.m. —Inaugural Reception Honoring President-Elect and Mrs. Alessi
Hosts—MSNJ and Bergen County Medical Society
All members, official guests, and their wives, and Auxiliary members are cordially invited
—Tickets $5.00 per person
8:00 p.m. —Inaugural Dinner
Hosts—MSNJ and Bergen County Medical Society
Music and Entertainment—Danny Davis and His Nashville Brass
Tickets $30.00 per person

Monday, May 14, 1979
7:00 a.m. —JEMPAC Breakfast
8:00 a.m. —Scientific Session on Orthopaedic Surgery, Family Practice
8:30 a.m. —Registration Opens
8:30 a.m. —Scientific Session on Surgery, Clinical Pathology
9:00 a.m. —Scientific Sessions:
Medicine, Rheumatism
Radiology
9:00 a.m. —Message Center Opens
9:00 a.m. —Scientific and Informational Exhibits Open
9:00 a.m. —JEMPAC Political Action Seminar
9:30 a.m. —Scientific Session on Chest Diseases
10:00 a.m. —Motion Picture Theatre
11:30 a.m. —Luncheon—NJ Society of Physical Medicine and Rehabilitation
12:00 noon —Luncheons:
NJ Chapter, American College of Chest Physicians
NJ Orthopaedic Society
1:00 p.m. —Scientific Sessions:
Gastroenterology and Proctology
Physical Medicine and Rehabilitation
1:00 p.m. —Meeting—NJ Foundation for Health Care
2:00 p.m. —Motion Picture Theatre
3:00 p.m. —House of Delegates
3:00 p.m. —Scientific and Informational Exhibits Close
6:00 p.m. —JEMPAC Wine and Cheese Reception

Tuesday, May 15, 1979
6:45 a.m. —Breakfast Meeting of the "Coalition of Southern NJ County Medical Societies"
Reservations: Mrs. Ruth E. Bunning, Exec. Sec., Burlington County Medical Society, P.O. Box 56, Medford, NJ 08055
7:15 a.m. —Breakfast Meeting—Bergen and Passaic County Medical Societies
Reservations: Mr. George Willis, Exec. Dir., Bergen County Medical Society, 170 Main St., Hackensack, NJ 07601
7:15 a.m. —Breakfast Meeting—Essex County Medical Society
Reservations: Mr. Arthur R. Ellenberger, Exec. Sec., Essex County Medical Society, 144 So. Harrison St., E. Orange, NJ 07018
8:30 a.m. —Registration Opens
9:00 a.m. —House of Delegates
12 noon —Registration Closes
2:00 p.m. —Annual Meeting—Board of Governors of the Medical Inter-Insurance Exchange of NJ
3:00 p.m. —Board of Trustees

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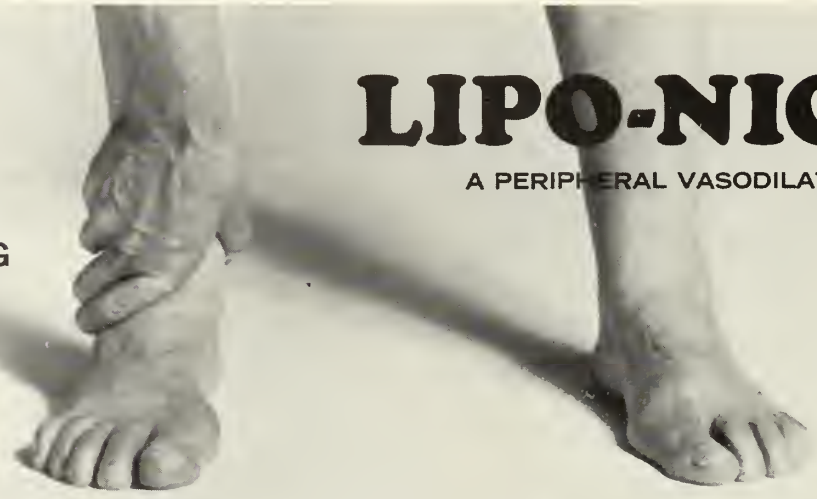
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DOSE: 1 to 5 tablets daily.
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Riboflavin (B-2) 2 mg.
Pyridoxina HCL (B-6) .. 10 mg.
DOSE: 1 to 3 tablets daily.
AVAILABLE: Bottles of 100, 500, 1000.

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Each time-release capsule contains:
Nicotinic Acid 300 mg.
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Thiamine HCL (H-1) .. 25 mg.
Riboflevin (B-2) 2 mg.
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In a special base of prolonged therapeutic effect.
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DOCTORS' NOTEBOOK

Trustees' Minutes December 17, 1978

A regular meeting of the Board of Trustees was held on Sunday, December 17, 1978, at the Executive Offices, Trenton. Detailed minutes are on file with the secretary of your county medical society. A summary of significant actions follow:

National Health Insurance . . . Approved the formation of an ad hoc committee to make recommendations and prepare a position statement on the national health insurance issue, to be composed of seven members: Chairman of the Board (Dr. Todd), President (Dr. Krueger), two members of the Board (Dr. Kovacs, who will serve as chairman, and Dr. Abrams), three AMA Delegates (Dr. Alessi, Dr. Benz, and Dr. D'Elia). There was considerable discussion of national health insurance at the December session of the AMA House of Delegates and a special session may be called in February; it is important that MSNJ have meaningful input in these deliberations.

MSNJ House of Delegates Special Session . . . Received a report* on the December 10th special session of the House of Delegates dealing with the subject of physicians' assistants which noted the following actions of the House:

1. Voted *not* to support the action of the Board of Trustees (10/15/78) to conditionally approve A-1387 and S-1192.

2. Voted to take the position of **active opposition** to A-1187 and S-1192 and to inform the legislature that licensure and/or registration of physicians' assistants will neither contribute to the quality nor decrease the cost of medical care.

3. Voted to adopt a plan* presented by Frank Y. Watson, M.D., Delegate from Essex County, indicating the role of the state society and the role of the county societies in the implementation of the above action.

Note: MSNJ staff has met with legislative consultants and it was suggested that a more effective approach would be to establish a statewide committee of presidents and presidents-elect of the counties who voiced the strongest opposition to the legislation, who would encourage the members of their respective counties to contact their State representatives. It was suggested that efforts be concentrated on retaining the bills in committee. The ultimate use of the keymen and the mass mailing to the Governor and legislators will await completion of the first stage of the plan as suggested by legislative consultants.

. . . Approved a recommendation that the Board of Trustees concur in the establishment of a statewide committee to consist of the presidents and presidents-elect of Bergen, Camden, Essex, Hudson, and Mercer County Medical Societies, who immediately will begin to encourage the members of their county societies to seek support among their State representatives for MSNJ's position of **active opposition to** A-1387 and S-1192.

Medicaid Resolution . . . Directed that the Executive Committee meet with the Medicaid Committee for the purpose of presenting an up-to-date report on the progress being made to carry out the intent of Resolution #22 (1978 House of Delegates) which urges physicians to treat but not bill for Medicaid patients thus putting Medicaid of New Jersey in noncompliance with federal regulations.

. . . Directed that a progress report on the Medicaid resolution be placed on the agenda for the Spring Conference of Presidents and Presidents-Elect, to keep them informed of developments.

Note: Meetings have been held with Mr. Pollock, Counsel to the Governor, and other members of the Governor's staff in an attempt to arrive at a practical solution to the implementation of the intent of the resolution. Pertinent data have been forwarded to Mr. Pollock and it is understood that the material is being processed through appropriate channels. The Executive Director advised the Board that he had received a letter from the Attorney General stating that if any two or more physicians undertake to implement the resolution as adopted by the House an antitrust violation suit will be instituted against MSNJ.

Cornerstone Club . . . Noted that contributions to date to the Cornerstone Club total $28,000.

CMDNJ Report . . . Received a report from Stanley S. Bergen, Jr., M.D., President of CMDNJ, which noted:

1. That consideration is being given to centralization of pediatrics at United Hospitals of Newark and transferring obstetrics to the College Hospital, which very likely would obviate the building of an inpatient psychiatric wing atop the Community Mental Health Center and thus free funds for staffing the perinatal center (see "CMDNJ Notes," page 136 this issue);

2. That tuition will be increased for 1979-1980 by $230 for those now paying $3,300 and by $280 for those who now pay $4,000.

3. That conflict continues over the decision as to whether John F. Kennedy Medical Center in Edison should be permitted to add beds in establishing a community mental health center for Edison-Metuchen-South Plainfield area. Rutgers Medical School Community Health Center is firmly established and believes that it can cover the area and extend these services to Cranbury, Plainfield, and South Brunswick if requested.

4. That the departments of medicine and obstetrics/gynecology are split between Raritan Valley and Middlesex General Hospitals; the department of

*See complete report of the session page 59, January 1979 issue of *The Journal.*

pediatrics is being transferred in its entirety to Middlesex General, along with the neonatal unit; the end-stage renal disease service should be moving to Middlesex General in the early spring, and the open-heart surgery department by July. Consideration still is being given to the future of Raritan Valley Hospital.

Hospices . . . Approved a recommendation from the Council on Public Relations (to whom Resolution #2, dealing with hospices—1978 House of Delegates —was referred) that the Board of Trustees request the joint executive committees of MSNJ and NJHA to discuss the development of hospices.

No-Fault Automobile Insurance—A-1286 . . . Voted to rescind its previous action of approving A-1286—Automobile No-Fault Insurance—and to take the position of **Conditionally Approved,** if it is amended by the sponsor to include the usual and customary fee schedule. (Currently the bill reads that fee schedules shall constitute maximum payment for insurers.)

Emergency Medical Services Regionalization Regulation . . . Supported the request of the Committee on Emergency Medical Services for a postponement of implementation of the proposed Emergency Medical Services Regionalization Regulation for the Designation of Emergency Medical Services Regions and Hospitals currently before the Health Care Administration Board —noting that the Emergency Medical Services Committee believes that the draft of the regulation submitted for publication in *The New Jersey Register* was premature. It lacks information concerning possible increased costs, a defined protocol as well as a geographic breakdown of EMS districts, and presently is voluntary on the part of the hospital to apply for the appointment of a resource, receiving, or associate hospital. The plan will fail if hospitals and squads do not participate. The Committee does not disagree with the document but believes that more information and interchange is needed before the regulation becomes effective.

ANNUAL MEETING
May 12-15

CMDNJ Notes*

Stanley S. Bergen, Jr., M.D.
President

By the time this column reaches print, the long-awaited move into the Newark Campus' new teaching hospital will have been completed. Out-patient services were moved from the CMDNJ-Martland Hospital to the new $78.9 million CMDNJ-College Hospital early in December. In-patient units were expected to have been relocated by the end of January.

The occupation of the CMDNJ-College Hospital marks the completion of the final phase of the College's $200 million construction program in Newark, a major step forward for health education, research, and service in New Jersey. The new facility will enhance CMDNJ's ability to offer superior health care, both to the Newark community, through its clinics and primary care services, and to the entire State, through its referral services in tertiary care specialties and its sophisticated equipment.

A plan to regionalize health services in the Newark area, which would result in significant savings for the State while enhancing the quality of care offered, is under consideration. The plan, which is currently under study by the College's board of trustees, as well as by state and regional health agencies, places all general pediatric and adolescent medicine in-patient care formerly located in Martland in the Children's Hospital Division of United Hospitals of Newark. United Hospitals, a CMDNJ affiliate, would consider closing its obstetric services, and these would be provided at College Hospital and other area hospitals. The transfer of pediatric services to United would provide space in the CMDNJ-College Hospital to accommodate Martland's psychiatric in-patient unit. As a result, CMDNJ would be able to table plans for building a new psychiatric unit, at a cost of about $2.5 million.

At our Central Jersey campus, the first step in the transition which will make Middlesex General Hospital the core teaching facility for the CMDNJ-Rutgers Medical School, has been completed. Three specialty services of obstetrics and gynecology have been relocated to the New Brunswick hospital. Currently in operation at Middlesex are: the gynecologic oncology service, reproductive endocrinology and infertility, and family planning.

Another major step in implementing the expanded affiliation between the Piscataway school and Middlesex General was the appointment of Dr. Gordon D. Benson as the hospital's chief of medicine. Dr. Benson, who also has been named professor of medicine and chief of the division of liver diseases at the medical school, will be responsible for directing educational programs in the department of medicine.

The transition program involves a $40-million expansion of the 362-bed hospital and construction of a medical education building by the College for faculty offices, classrooms, laboratories, and a library. The entire transition is expected to take at least three years. The next major move will be pediatric services, slated for early this year.

The expanded affiliation with Middlesex will allow the CMDNJ-Rutgers Medical School to continue increasing its third and fourth year classes. When the College acquired CMDNJ-Raritan Valley Hospital in 1970, the Piscataway school just was beginning its transition to a full-service medical school from a two-year basic science institution. Since the Green Brook facility can support clinical training for a maximum of only 56 students, nearly half of each entering class had to leave the State to get their M.D. degrees. With Middlesex General as its core teaching facility, CMDNJ-Rutgers Medical School will be able to increase third and fourth year classes to 108 students each. The affiliation, which was signed last year, already has become productive for the medical school. The current senior class has been expanded to 79 students; the current junior class has grown to 91.

Another factor in the medical school's growth has been the close affiliation with Cooper Medical Center, Camden. The hospital has been identified as the core teaching facility of the College's South Jersey Medical Education Program, which places students of CMDNJ-Rutgers Medical School in South Jersey hospitals for their clinical training. The first group of 12 third-year students began studies at Cooper last September.

The question of the ultimate utilization of the CMDNJ-Raritan Valley Hospital is receiving serious consideration at CMDNJ. The 131-bed unit will, of course, remain a teaching facility of the medical school during transition to Middlesex General. Long-term options now are being determined through a study being conducted by Coopers &

Lybrand, a national consulting firm. The study will embrace all facets of Raritan Valley's operations, including a review and projection of the area population's health care needs. It will generate recommendations as to how the facility best can serve the community in the future.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Office of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ALLERGY/IMMUNOLOGY—Alan R. Green, M.D., 1315 Ethel Street, Glendale, CA 91207. New York Medical 1971. Partner, associate in single-specialty group, or purchase of practice. Available August 1979.

ANESTHESIOLOGY—Parvin Javadi, M.D., 57 Montague Street, Apt. 9G, Brooklyn, New York 11201. Shiraz (Iran) 1964. Board eligible. Group, solo, partnership. Available.

Jitendra G. Patel, M.D., 133-52 Avery Avenue, Flushing, New York 11355. M. P. Shah (India) 1973. Board eligible. Any type practice. Available July 1979.

CARDIOLOGY—Michael M. Neumann, M.D., 5415 North Sheridan Road, Chicago, IL 60640. Dusseldorf (Germany) 1971. Subspecialty, internal medicine. Board certified (IM). Group or institution. Available.

Dominick J. Passalacqua, M.D., 2392 East 5th Street, Brooklyn, New York 11223. Bologna (Italy) 1972. Cardiac laboratory for invasive and non-invasive procedures. Available January 1979.

Michael J. Zema, M.D., 23 Rosemont Avenue, Elmwood Park, New Jersey 07407. Cornell 1974. Board certified—IM. Board eligible—Cardiology. Group, partnership, solo (with or without part-time institution). Available July 1979.

Muljibhai J. Thakkar, M.D., 255-25 75th Avenue, Glen Oaks, New York 11004. Baroda (India) 1972. Solo. Available July 1979.

FAMILY PRACTICE—Jack A. Devor, M.D., 828 Cape View Drive, Fort Myers, Florida 33907. Hahnemann 1966. College or university health service. Available.

Elmer S. Gilo, M.D., St. Joseph Hospital, Stamford, CT 06904. University of the East (Philippines) 1973. Board eligible. Group, solo, partnership; also teaching. Available July 1979.

Charles A. Joseph, M.D., 99 Morris Avenue, Summit 07901. CMDNJ 1976. Board eligible. Group or partnership. Available July 1979.

Rosario A. Bascon, M.D., 15 St. Andrews Place, Apt. 2-A, Yonkers, NY 10705. Cebu (Philippines) 1967. Board certified. Solo, partnership, group. Available.

HEMATOLOGY—Jagmohan K. Kalra, M.D., 2 Robin Lane, Plainview, N.Y. 11803. Lady Hardinge (India) 1970. Subspecialty, oncology. Board certified (IM) (Hematology). Group, partnership, hospital. Available July 1979.

INTERNAL MEDICINE—Peter K. W. Chak, M.D., 200 Bethel Loop, Apt. 20D, Starrett City, Brooklyn, New York 11239. Hong Kong 1970. Board eligible. Group, partnership, associate, solo. Available June 1979.

Robert S. Goldblatt, M.D., 150 Division Street, Derby, Connecticut 06418. George Washington 1974. Subspecialty, gastroenterology. Board eligible. Group, partnership. Available July 1979.

Alan M. Nelson, M.D., 41B Monroe Street, Milford, Connecticut 06460. Georgetown 1974. Subspecialty, gastroenterology. Board certified (IM). Group, partnership. Available July 1979.

Vithal Kusuma, M.D., 1227 South Harlem, #504, Berwyn, Illinois 60402. Osmania (India) 1970. Subspecialty, gastroenterology. Board certified (IM). Hospital, group, solo. Available July 1979.

Steven Nussbaum, M.D., 891 Clopper Rd., Apt. B-1, Gaithersburg, Maryland 20760. SUNY—Downstate, 1974. Subspecialty, gastroenterology. Board certified (IM). Group, partnership. Available July 1979.

Stephen A. Atlas, M.D., 159 Hawthorne Avenue, Central Islip, NY 11722. Wisconsin. Board eligible. Group or partnership. Available July 1979.

Yugal K. Maheshwari, M.D., 10555 Fondren, Apt. #727, Houston, Texas. J. N. Medical College (India) 1970. Subspecialty, endocrinology. Board eligible. Partnership, associate, single or multi-specialty group. Available July 1979.

Michael D. Burday, M.D., 139-1 Kirkbride Road, Voorhees 08043. Georgetown 1976. Group or partnership. Available July 1979.

Ravi K. Malpani, M.D., 1165 Route 22, Apt. 22, North Plainfield 07061. Osmania (India) 1972. Subspecialty, pulmonary medicine. Board eligible. Group, solo, partnership, or hospital-based. Available July 1979.

John Kassabian, M.D., 25 Burnett Terrace, West Orange 07052. CMDNJ 1974. Subspecialty, pulmonary medicine. Board certified. Any type practice. Available July 1979.

Jerome W. Takiff, M.D., 2917 E. Turquoise, Phoenix, Arizona 85028. Cornell 1973. Board certified. Group. Available.

Kenneth Lipkowitz, M.D., 76 E. Park St., Apt. 26, East Orange, N.J. 07017, Guadalajara 1974. Board eligible. Partnership or group. Available July 1979.

N. M. Koshy, M.D., 8 Slate Creek Dr., Apt. 9, Cheektowaga, New York 14227. Kottayam-Kerala (India) 1971. Board certified. Subspecialty, cardiology. Group, partnership, solo, institution. Available July 1979.

Kiril Desai, 108-18 63rd Street, Forest Hills, New York 11375. Baroda (India). Subspecialty, cardiology. Group, partnership, salaried position. Available.

Vennaralagappan Mahadev, M.D., 429 Case Street, Kingsford, Michigan 49801. Madurai (India) 1967. Board certified. Subspecialty, cardiology. Group or solo. Available August 1979.

Harvey M. Cohen, M.D., 8 Deerwood West, Apt. 44C, Irvine, CA 92714. Syracuse 1972. Board certified. Subspecialty, gastroenterology. Solo, group, partnership. Available.

Taha Hamoui, M.D., 9831 Timerwood Circle, Louisville, KY 40223. Damascus (Syria). Board certified. Subspecialty, gastroenterology. Group, partnership, solo. Available July 1979.

NUCLEAR MEDICINE—R. Murty Krishnamsetty, M.D., 101 West 15th Street, Apt. 4-E North, New York, NY 10011. Banaras H. (India) 1971. Special interest, therapeutic radiology. Board eligible. Board certified in T.R. Group or partnership. Available July 1979.

OBSTETRICS/GYNECOLOGY—Khatoon T. Ginwala, M.D., 1817 Rolling Lane, Cherry Hill 08003. Hahnemann. Board eligible. Available July 1979.

Nilima Parekhji, M.D., 12000 Fairhill Road, Apt. 814, Cleveland, Ohio 44120. T.N. Medical (India) 1972. Board eligible. Group, partnership, solo. Available July 1979.

Jeffrey M. Reinkraut, M.D., 40-01 Little Neck Pkwy., Little Neck, NY 11363. Rutgers (CMDNJ) 1975. Board eligible. Group or partnership. Available July 1979.

OPHTHALMOLOGY—Zdenka F. Macek, M.D., 271 Nandina Terrace, Philadelphia 19116. Charles University (Czechoslovakia) 1966. Board eligible. Group, partnership, association, institution. Available.

PEDIATRICS—R. P. Vasani, M.D., 1575 Boston Ave., #C-12, Bridgeport, Connecticut 06610. M.S. University (India) 1974. Board eligible. Group or partnership. July 1979, or sooner.

Mehboob A. Sachani, M.D., 1 Slate Creek Rd., Apt.3, Cheektowaga, New York 14227. Seth G. S. Medical (India) 1974. Board eligible. Any type of practice. Available July 1979.

C. V. R. N. Reddy, M.D., 57 Rupert Ave., Staten Island, NY 10314. Kakatiya (India) 1968. Board eligible. Group, partnership, solo. Available.

Theodore Sher, M.D., 170 West End Avenue, New York, NY 10023. SUNY—Downstate 1974. Special interest, pediatric allergy. Board eligible. Group or partnership. Available July 1979.

Warren P. Silberstein, M.D., 17 Bartlett Drive, Middletown, NY 10940. SUNY—Downstate. Board eligible. Group practice. Available July 1979.

Vijaya Lakshmi Malpani, M.D., 1165 Route 22, Apt. 22, North Plainfield 07061. Osmania (India) 1972. Board eligible. Group, solo, hospital-based, partnership. Available July 1979.

Bart Asner, M.D., 3949 Los Feliz Blvd., Los Angeles, CA 90027. Pennsylvania 1976. Group or partnership. Available July 1979.

Cecil F. Desai, M.D., 2112 Starling Ave., #5K, Bronx, NY 10462. B. J. Medical College (India) 1968. Board eligible. Salaried position. Available July 1979.

M. S. Krishna, M.D., 32127 Concord Dr., Bldg. 6, Apt. F, Madison Heights, Michigan 48071. Bangalore (India) 1971. Board eligible. Group or partnership. Available July 1979.

PSYCHIATRY—Elaine Nicola, M.D., 117 Grove Street, Ramsey 07446. Northwestern 1970. Board certified. Group, partnership, or clinic. Available February 1979.

PULMONARY DISEASES—Peter Y. Lee, M.D., 319 East 24th Street, Apt. 21D, New York 10010. NYU 1974. Board certified (IM). Board eligible (pulmonary diseases). Group, institution, association. Available July 1979.

Shreedhar B. Shetty, M.D., 116B Davey Street, Bloomfield 07003. Kasturbha (India) 1968. Board eligible. Group. Available July 1979.

RADIOLOGY—Pongthep Thepsongwajja, M.D., 2309 NW 38th Street, Apt. 75, Lawton, Oklahoma 73505. Siriraj (Thailand) 1972. Board certified. Group, partnership, solo. Available.

R. Murty Krishnamsetty, M.D., 101 West 15th Street, Apt. 4-E North, New York, NY 10011. Banaras H. (India) 1971. Subspecialty, nuclear medicine. Board certified (therapeutic radiology). Board eligible (N.M.) Group, partnership. Available July 1979.

SURGERY, GENERAL—Michael J. Kassouf, M.D., 4 Dartmouth Ave., Apt. 3A, Bridgewater, New Jersey 08807. French Faculty (Beirut, Lebanon) 1970. Board eligible. Group, partnership, solo. Available September 1979.

William A. Schuckardt, Jr., M.D., 13500 Shaker Road, Cleveland, Ohio 44120. Case Western Reserve 1974. Board eligible. Group, solo, or partnership. Available July 1979.

John L. DeBoer, M.D., 1242 B Cedar Street, Fort Dix, Vermont 1971. Board certified. Single-specialty group, partnership, multi-specialty group, solo. Available June 1979.

David W. Fricke, M.D., 1020 Orr's Bridge Road, Mechanicsburg, PA 17055. University of Pennsylvania 1960. Board certified. Any type, partnership or group practice preferred. Available.

M. Y. Dhar, M.D., 318 East 15th Street, Apt. 8-M, New York, NY 10003. G. R. Medical College (India). Board eligible. Any type practice. Available July 1979.

Stuart A. Engel, M.D., 545 39th Avenue, San Francisco, CA 94121. Johns Hopkins 1974. Group or partnership. Available August 1979.

Michael F. Lane, M.D., 7901 Henry Avenue, Apt. C-411, Philadelphia, PA 19128. SUNY—Downstate 1973. Board eligible. Group or partnership. Available July 1979.

Donald F. Temple, M.D., 44 Morningside Dr., Apt 5, New York 10025. Cornell 1974. Board eligible. Group, partnership, hospital, solo. Available July 1979.

SURGERY, ORTHOPEDIC—Ronald B. Greene, M.D., 1405 Bay Drive, Miami Beach, FL 33141. Temple 1972. Board eligible. Solo, group, or association. Available July 1979.

Lawrence Schweitzer, M.D., 23 Woodward Crescent, Halifax, Nova Scotia B3M1J6. Louvain 1975/Dalhousie 1979. Board eligible. Group, partnership, solo. Available July 1979.

T. Ghavamian, M.D., P.O. Box 3452, San Diego, CA 92103. Teheran 1953. Board certified. Group, partnership, solo, academic. Available.

SURGERY, THORACIC—Chukuma I. Okadigwe, M.D., 590 Flatbush Avenue, Apt. 14-P, Brooklyn, NY 11225. Colorado 1968. Board eligible. Group, partnership, solo. Available.

UROLOGY—Howard B. Somers, M.D., 1153 East 72nd Street, Brooklyn, New York 11234. Guadalajara 1973. Board eligible. Partnership, group, institution. Available July 1979.

Arnold M. Grebler, M.D., 19 Spartan Avenue, Staten Island, NY 10202. Bologna (Italy) 1974. Partnership or group. Available July 1979.

Stewart L. Feldman, M.D., 34 Hunter Circle, Mount Holly 08060. Vermont 1971. Board eligible (general surgery). Group, partnership. Available July 1979.

Ekiong C. Tan, M.D., 8478 Ohern Street, Omaha, Nebraska 68127. Santo Tomas (Philippines) 1970. Board eligible. Group or partnership. Available July 1979.

Daniel H. Piazza, M.D., 741 Peninsula Court, Ann Arbor, Michigan 48105. Illinois 1972. Board eligible. Partnership, single or multi-specialty group. Available July 1979.

Gerald A. Goldman, M.D., 2913 Willowwood Drive, Erie, PA 16500. Guadalajara 1972. Board eligible. Association leading to partnership. Available July 1979.

Report from the Foundation

Daniel J. O'Regan, M.D.
Medical Director

The year 1979 promises to be an interesting one. Attention will be attracted to the Department of Health as it starts to implement its prospective rate-setting program for hospitalized patients. A federal contract was awarded to the department in December, to establish a new system that will be based on costs of treatment and severity of illness (case-mix), instead of flat per diem rates. This will bring the Diagnosis Related Groups (DRGs) into the picture. Another part of this venture was the passage last year of S-446, which provides for a rate setting commission to oversee the operation of the system. No predictions can be made on how effective this type of payment system will be. Twenty-two hospitals are expected to begin trying the method as of this month. The New Jersey Register will be required reading, as regulations are proposed to implement the legislation. One of the first jobs will be to address the problem of the medically indigent. S-446 provides for dividing the costs of the hospitalized indigents among *all* payers. This will affect many of our inner-city institutions. This could have a profound effect on the way hospital care is paid for. There also will be quality screens built into the data system. Cases which fall outside the established parameters will be referred to the respective PSROs for peer review.

Phencyclidine: A Review

MATERIAL

Phencyclidine, commonly called PCP, in its pharmaceutically pure form is a white powder which readily dissolves in water. In "street" form PCP often is adulterated and misrepresented as other drugs. It is highly variable in appearance and is sold in powder and tablet form, both in many colors. Often it is placed on parsley or on other leaf mixtures to be smoked as cigarettes (joints). When misrepresented, it is most commonly sold as THC (the principal psychoactive ingredient in marihuana, which in reality is rarely, if ever, available on the street). But phencyclidine also has been sold as cannabinol (another marihuana constituent), mescaline, psilocybin, LSD, and even as amphetamine or cocaine. Because of the variability in street names and its frequent misrepresentation, the user may be unaware of what he or she has ingested or may be mistaken about its true identity.

Some of the street names for phencyclidine include: angel dust, dust, crystal, cyclones, embalming fluid, elephant or horse tranquilizer, killer weed, superweed, mintweed, mist, monkey dust, PeaCe Pill, rocket fuel, goon, surfer, KW, and scuffle. The wide range of regional names for this illicit drug, as well as variations in its appearance, mean that the casual user may not be aware that he or she has used PCP or may fail to report its use in surveys because he or she does not know the drug by the survey designation. A further complication in user identification of PCP is the fact that phencyclidine is sometimes also used along with such other drugs as barbiturates, heroin, cocaine, amphetamine, methaqualone, LSD, mescaline, and procaine.

Depending on the care with which the synthesis of PCP is carried out, the street drug may contain a variety of impurities, including potassium cyanide. While a mixture of marihuana and PCP has been thought to be common, in reality this combination rarely has been reported by street drug analysis laboratories. At least one major laboratory on the west coast which does such drug analysis never has encountered the combination.

Significantly adding to the risk of PCP use, especially when it is taken orally, is the wide variability in purity of the street drug. Even when it is not misrepresented, the percentage of PCP contained has been found to be quite variable. Generally, samples represented as "crystal" or "angel dust" have tended to be purer than those sold under other names or misrepresented as other drugs.

In addition to phencyclidine, there are over thirty chemically similar analogs, some of which are capable of producing similar psychic effects. These also can be synthesized with varying degrees of difficulty, and some have already appeared on the street. Thus the problem of "tracking" the use of PCP and related materials is unusually difficult.

Phencyclidine can be synthesized relatively easily. The "starting" chemicals are widely available and do not lend themselves to more stringent controls. While some of the mass media accounts have exaggerated the ease with which phencyclidine can be made, it is not particularly difficult and can be done by individuals with only modest technical training and without elaborate equipment.

The precise classification of PCP presently is unsettled. PCP has stimulant, depressant, hallucinogenic, and analgesic properties, some of which are dose-dependent. One proposed classification is with ketamine as a "dissociative anesthetic."

Phencyclidine is used legally in veterinary medicine as an animal immobilizing agent. Although it was originally developed as an anesthetic for use with humans, it was later abandoned for the purpose because in some patients it produced post-operative thought disturbances and agitation. PCP made its first illicit appearance in 1965—on the west coast. At that time it rapidly developed a bad street reputation and had only limited popularity. Since then there have been sporadic outbreaks of its use. Most recently there is evidence from a variety of sources suggesting a marked increase in use (cf. Extent of Phencyclidine Abuse).

Between 1971 and 1974 street samples of phencyclidine analyzed on the west coast were found to be mixed with other drugs in about half of the samples tested. In samples tested since 1975, however, only half as many (25 percent of the total) were found to contain additional drugs. Still more recently, PCP or one of its analogs (TCP) has constituted 10 percent of all samples analyzed by this Los Angeles laboratory. Only one in thirty-three of the samples which contained PCP actually was sold as such.

When PCP is sold as a granular powder ("angel dust"), it is usually relatively pure, consisting of perhaps 50 to 100 percent phencyclidine. However, sold under a variety of other names and in other guises, the purity is from 10 to 30 percent with leafy mixtures generally containing still smaller amounts of the drug.

Although originally ingested orally, presently PCP is commonly smoked or snorted. Intravenous use is less frequent, but has been reported. By smoking, the experienced user is better able to limit the dose to a level with which he or she is comfortable and is perhaps less likely to overdose. When the drug is taken by mouth, there is a longer period before the drug takes effect, and the dose is usually greater, resulting in more common adverse reactions (overdose) by this route.

EXTENT OF ABUSE

Attempts at estimating the extent of PCP use are fraught with difficulty. Since the drug often is sold as any one of a variety of other psychoactive substances, the user, especially if he or she is inexperienced, may not realize PCP is involved. The fact that phencyclidine is known by an unusually large number of street names and varies widely in its physical appearance also contributes to confusion in casual user identification. Most official reports of drug use have in the past lumped PCP with other hallucinogens, making trend detection from such sources difficult. Moreover, the patient admitted for emergency treatment of PCP-induced bizarre behavior is likely to be diagnosed as acutely schizophrenic rather than as a toxic drug reaction. Patients admitted with injuries from automobile accidents, fires, and drownings in which PCP was a cause may not be so identified. Thus a marked increase in use and in use-related drug emergencies may not be reflected in emergency room statistics.

Despite these limitations, there are a number of converging lines of evidence suggesting increased use in recent months. These range from reports of law enforcement agencies on disruption of illegal laboratory production to self-reports by users. While available survey data have definite limitations—for example, the experimental user may not realize he has in fact used PCP—it provides a minimal estimate of the level of phencyclidine use as well as some short-term trend indicators. In NIDA's 1976 National Survey, three percent of youth between 12 and 17 years of age at the

*This study was prepared by Robert C. Petersen, Ph.D., and Richard C. Stillman, M.D., of the National Institute on Drug Abuse, 5600 Fishers Lane, Rockville, Maryland 20850, and is reprinted with permission.

time of the survey acknowledged having used PCP at some time in their lives. Among young adults 18 to 25, the age category in which virtually all drug use peaks, nearly one in ten reported having used PCP prior to the 1976 survey. By contrast, data from the 1977 National Survey suggests that the percentage who have ever used in the 12 to 17 year-old group has nearly doubled since the earlier survey (5.8 percent vs. 3.0 percent in 1976). Use by the 18 to 25 age group also has increased markedly, from 9.5 to 13.9 percent. The likelihood that these increases were simply the result of year sampling variation was less than one in a hundred.

Some indication of the extent to which a drug poses serious problems is provided by the government's Drug Abuse Warning Network (DAWN). DAWN is a national reporting system which collects reports of drug-related deaths and drug-related emergencies involving hospital emergency room treatment. For reasons that already have been outlined, such figures are likely to be minimal estimates of the PCP problem. They are, however, useful in providing some indication of trends.

When data are examined from the 602 emergency rooms which consistently reported to DAWN from November 1974 to October 1976, the rate of PCP emergencies doubled during that period (there were 111 mentions in October 1976 contrasted with 54 in October 1974). Reports of PCP-related deaths show nearly as great an increase when the period from April 1976 to March 1977 is contrasted with that of the previous year (30 PCP-related deaths vs. 17 the year before). Again, such figures are likely to be minimal estimates of the actual extent of PCP-related mortality.

A major reported cause of PCP deaths in California has been drowning. In one series of 19 PCP deaths investigated in two California counties, 11 of the 19 were from drowning—one while in the shower. The PCP user readily loses his orientation while swimming or immersed and frequently drowns, sometimes in very small amounts of water. Unless PCP in the body is specifically searched for under such circumstances, it is very likely to be missed. Similarly, deaths resulting from violent behavior and accidental deaths resulting from PCP-intoxication may not be reported as PCP-related. Suicide while under the influence of the drug was the cause of death of three additional persons in the California study. Another death was the result of threatening behavior leading to the individual's being shot (Burns and Lerner 1978).

Another source of data which provides some indication of the extent of phencyclidine use among chronically drug-using youth is the National Youth Polydrug Study (NYPS), in which 97 drug abuse treatment programs specializing in treating youthful drug abusers participated. During the period from September 1976 to March 1977 interviews were conducted collecting detailed data on patterns of drug abuse for 2750 new clients under age 19. Within this sample nearly a third (31.8 percent) reported having ever used PCP. It was used more often than inhalants, sedatives, cocaine, and opiates other than heroin and methadone. Females in the sample were as likely to have used PCP as males. Whites were far more likely to report using it than either black or Hispanic clients (42.3 percent of whites, 8.5 percent of blacks and 9.0 percent of Hispanics). Females in the sample were found to have used PCP first at a slightly earlier age (14.4 years) than males (14.7 years). American Indians began use at the earliest age (13.9), followed by Hispanics (14.0), whites (14.6) and blacks (15.3). Two-thirds of those who reported ever having used PCP reported continued use at a rate of at least once a week for at least one month. PCP users also had used twice as many other recreational drugs as had non-PCP users (6.0 vs. 2.8 substances respectively). Half the current PCP users were using PCP once a week or more often during the three-month period that was studied. While this data-source is obviously not representative of youth generally, it does suggest that PCP use is quite common among abusers of other drugs.

OCCASIONAL AND CHRONIC USE OF PCP

Effects of Acute Intoxication—The best known effects of PCP are so unpleasant that many have wondered how PCP possibly could prove popular. Many one-time users will not knowingly use it again. Yet others enjoy it consistently and use it chronically.

Depersonalization and distortion of body image are among the effects reported most frequently from a moderate amount of PCP. The user feels a sense of distance and estrangement from his surroundings. Time expands and body movements are slowed. Muscular coordination worsens as proprioceptive impulses are dulled; the user may stagger as though drunk. A sense of increased physical strength frequently occurs. Speech is blocked, sparse, and purposeless. Auditory and sometimes visual hallucinations may occur, more frequently at higher doses, and feelings of severe anxiety, impending doom, or death may appear and disappear. Touch and pain sensations are dulled. Bizarre behavior, such as nudity in public places, has been reported.

Feelings of immobility, numbness, and detachment may be desirable in themselves to some users. Perhaps easier to understand is the attractiveness of some of the other effects reported by many PCP users: feelings of strength, power, and invulnerability, coexisting with dreamy estrangement and comical lack of coordination. The drug is described as stronger than marihuana, more comparable to LSD, but basically "in a class by itself." As with these other drugs, a few users spoke of distinctly new perspectives and of seeing their lives freshly and with a sense of unity. Some users reported distinct euphoria or marked depression with the drug. Great variability exists in effects from one user to the next.

Chronic Use—Modes of Use—The novice user often is introduced to the drug openly or covertly by smoking it sprinkled on marihuana or parsley leaves in a "joint" in amounts varying substantially from 1 to 100 mg each. Occasionally it is snorted, or taken by mouth in capsules or pills. Chronic users might take anywhere from 100 mg to 1 gram in a 24-hour period. Intravenous use has been reported and is less common, but by no means rare. The effects of each route of ingestion are essentially similar, differing in the rapidity of onset, but lasting 4 to 6 hours, with an even longer "coming down" period. Accounts from users suggest some tolerance, with increasing doses needed at the end of a "run" to achieve the same effects. PCP very frequently is used as a social drug, taken in groups of users rather than alone.

The Chronic User—The premorbid personalities of PCP abusers are not consistently described in the literature. PCP users appear to have a variety of premorbid personalities, not fitting a single pattern. Fauman and Fauman (1977) made the point that many of those who become psychotic with this drug appear to resemble persons who become psychotic using LSD. However, PCP abusers include not only socially marginal people, but also people of substantial achievement without obvious premorbid psychopathology.

Effects of Chronic Use—For chronic PCP users, PCP is in fact a drug of choice. Burns and Lerner (1978) report that chronic phencyclidine users experienced persistent cognitive and memory problems. Speech difficulties included stuttering, poor speech articulation, and difficulties in expressing themselves to others, effects lasting six months to a year following prolonged daily PCP use. Mood disorders also occurred; viz., depression, anxiety, and violent behavior. Purposive activity became more difficult with resultant loss of employment or impaired school performance. Chronic users have reported losing 10 to 35 pounds of body weight during regular use. Following a 2 to 3 day "run," users need great amounts of sleep and may awaken feeling disoriented and depressed. In later stages of chronic use, outright paranoid and violent behavior with auditory hallucinations often appeared.

The possible role of PCP in precipitating long-term psychosis is at present poorly understood. It may be that persistent psychosis occurs in individuals who are latently schizophrenic as a result of some "triggering" function of the phencyclidine. Whatever the cause, there is some clinical evidence that individuals who have used PCP in the past may later develop more persistent schizophrenia despite some months of abstinence from the drug.

The fact of having used PCP repeatedly without serious adverse effect is apparently no guarantee that more serious consequences will not occur unexpectedly on a given occasion of use. At least one group of clinical investigators (Burns and Lerner 1978) believes that if use is persistent, ultimately adverse consequences are virtually inevitable.

Treatment of Chronic PCP Use—As with other drug abuse, the motivations of the user are crucially important in the approach to treatment. In at least one geographic area of high PCP abuse, a downturn in emergency cases, possibly indicating less widespread general use, followed an intensive effort by the local community mental health center to publicize the adverse effects of the drug. Unfortunately, numerous case accounts suggest that the user who consistently enjoys PCP will be reluctant to give up its use, since it is relatively cheap, easily available, and quite powerful. Although for many it is a particularly disagreeable drug, for others it is quite enjoyable. Whether this reflects a psychopharmacologic difference between these groups is not known, but its genuine appeal to some must be taken into account in any therapeutic or educational program. Not only is the drug readily available, but so is the example of users who consistently enjoy it. So, also, is the example of users who experience severe depression while taking the drug, but who continue their use of it notwithstanding.

PCP OVERDOSE

Diagnosis—The victim of an overdose of PCP may exhibit many different symptoms during the course of his intoxication, depending on the dose, how recently it has been ingested, and frequency of prior use. Some cardinal signs of PCP overdose are: ataxia, vertical and horizontal nystagmus, assaultiveness or catatonic staring, and generalized anesthesia. Severe convulsions may also occur in extremely heavy overdoses, and PCP exhibits the lethal properties of a central nervous system depressant, with respiratory depression, coma, and death.

The dose of PCP which may result in a trip to an emergency room is as varied as the presenting symptoms. A single cigarette may, in the inexperienced (or deceived) user, produce overwhelming feelings of acute anxiety requiring prompt but relatively brief reassurance and therapeutic support. In adults, quantities in excess of a half a gram have been used and it is particularly in this dose range (or higher) that life-threatening effects are likely to be manifested along with severe behavioral disturbance.

The diagnosis of PCP overdosage is frequently missed (Fauman and Fauman 1977). Because the presenting symptoms so often closely resemble those of an acute schizophrenic episode, this is a common misdiagnosis. Aronow and Done provide a useful summary (Aronow and Done 1978) of the range of physical and psychological symptoms likely to be encountered in the emergency room situation.

At relatively low doses (on the order of 5 mg in the adult leading to an estimated serum level of 20-30 ng/ml) they describe the patient as likely to show some of the following symptoms:

agitation and excitement
gross incoordination
blank stare appearance
catatonic rigidity
catalepsy
inability to speak
horizontal or vertical nystagmus
loss of response to pinprick
flushing
diaphoresis
hyperacousis

Psychologically the patient may have the following subjective responses: changes in body image, estrangement, disorganization of thought, feelings of inebriation, drowsiness, and apathy. The patient may show marked negativism and hostility as well as bizarre behavior. Later, the patient may be amnesic for the drug episode.

At moderate doses (approximately 5-10 mg., leading in the adult to serum 30-100 ng/ml serum level), Aronow and Done (1978) describe the following symptom picture:

coma or stupor
eyes remain open
pupils in midposition and reactive
nystagmus
vomiting
hypersalivation
repetitive motor movements
myoclonus (shivering)
muscle rigidity on stimulation
flushing
diaphoresis
fever
decreased peripheral sensations (pain, touch, and position)

At still higher doses (over 10 mg. leading, in the adult, to serum levels of 100 ng/ml and higher) the authors describe the following:

prolonged coma (from 12 hours to days)
eyes closed
variable pupil size, but reactive
hypertension
opisthotonic posturing
decerebrate positioning
repetitive motor movements
muscular rigidity
convulsions (at doses of 0.5 to 1 mg/kg)
absent peripheral sensation
decreased or absent gag and corneal reflexes
diaphoresis
hypersalivation
flushing
fever

(The doses cited are rough guidelines; other investigators suggest that significantly higher doses of PCP are needed to produce each of these clinical pictures.)

If the symptoms found are suggestive of recent PCP ingestion, a urine or blood sample may reveal the presence of the drug. If a large amount has been ingested, considerable quantities of PCP will be present in urine for several days (or more). Attempts should be made to elicit a drug history from informants, although it is quite possible that they may only know the drug under one of its street names (cf. Material) or may not

even know that the patient has taken phencyclidine if the drug has been misrepresented. Since the drug often renders the patient amnesic for the circumstances of taking it, he or she may not remember having taken it or may only recall it during a later period of recovery. A history of recent ingestion of other drugs may complicate the diagnosis and treatment. Thus the diagnosis may sometimes be established by history, sometimes by laboratory tests, by the physical and psychological course of the patient, or by some combination of these.

Treatment—Life-support Measures—The hospital management of the PCP overdose victim is divided into phases corresponding to the time course of action of the drug. The initial phase requires symptomatic, often intensive, medical management. It includes the most acute physical effects of the drug, including the possibility of respiratory depression, convulsions, and coma. Treatment of this phase requires full life-support capabilities. Acidification of the urine (see below) will markedly hasten the excretion of PCP. Since there is no specific antagonist for PCP, other treatments are symptomatic. PCP often induces hypersecretion in the pharnyx, which must be suctioned. Respiratory depression calls for external respiratory assistance. Hyperthermia can be treated with external cooling. Life-threatening consequences of rapid development of hypertension can be treated with diazoxide (Hyperstat®) or hydralazine hydrochloride (Apresoline®). Convulsions can be managed with IV diazepam and constant attention to maintenance of an unimpaired airway. However, some feel diazepam may prolong the course of the intoxication.

Isolation—Most clinicians advocate placing the patient in an isolated environment during this period, reducing sensory stimulation as much as possible in order to minimize the phase of excitability, irritability, anxiety, paranoia and violence which often follows the obtunded or comatose phase. Needless to say, such isolation of the patient (in quiet rooms) cannot be at the expense of vigilant medical monitoring of vital signs and response to emergency life-support management. Patients often are so unmanageable that restraints are necessary, and the help of four or five (not one) burly aides will be needed to prevent injury to staff or patients. If it is necessary to administer tranquilizing agents to keep the patient manageable, do not use phenothiazines during this acute stage because of the anticholinergic potentiation with PCP. Haldol® (haloperidol) has been recommended (5 mg IM repeated hourly until the patient is under control).

Detoxification—Recently ingested drugs should be removed by gastric lavage. Done (1978) describes an emergency detoxification procedure which achieves urine concentrations of PCP which are much higher than in blood by lowering the urine pH to 5 or less. They recommend administration of ammonium chloride by gastric intubation or, in severe cases, intravenously. (Prior screening must be done for the concomitant presence of such other drugs as phenobarbital or salicylates, whose excretion might be adversely affected by acidification, as well as by liver damage.) To acidify by intubation they use 2.75 mEq/kg ammonium chloride in 60cc of saline solution every 6 hours along with intravenous ascorbic acid (2 gm/500 cc IV fluid q 6 hours) until the urine pH drops below 5. For patients in deep coma, they advise using intravenous ammonium chloride (2.75 mEq/kg as a 1-2 percent solution in saline) with precise and repeated monitoring of blood pH, blood gases, BUN, blood ammonia levels, and electrolytes.

The treatment of acute toxic reactions usually belongs in an intensive care unit, not an emergency room. Constant gastric suctioning is essential and treatment should be continued past the point of awakening from coma. The excretion of PCP is variable and prolonged. Ten days to two weeks of further acidification using cranberry juice is indicated, because of remaining body stores of PCP.

If medical treatment is successful, the patient's coma will lighten, only to be replaced, frequently, by delirium, paranoia, and violent assaultiveness. This may occur at the same time as life-support measures are still necessary, requiring constant attention from the medical staff so that intubation and intravenous lines remain open and intact. Any patient in whom coma is followed by a prolonged period of confusion should, as recommended by Burns and Lerner (1978) have a psychiatric consultation prior to discharge to evaluate residual paranoia or potentially suicidal depression.

PCP PSYCHOSIS

a. Diagnosis—Some patients experience a psychotic phase which lasts from several days to several weeks despite abstinence from further PCP. The psychosis may occur after single doses of PCP. Luisada and Brown (1976) have divided these psychoses into three stages: the initial stage, characterized by violent psychotic behavior, lasting perhaps five days; the second stage, with restless but more controlled behavior; and the final stage, including rapid improvement of thought disorders and amelioration of paronoia.

It should be emphasized that this psychotic stage may be the first stage at which the patient enters treatment, often through a psychiatric hospital emergency room rather than a general medical hospital emergency service. He may or may not have passed through an earlier stage of reduced consciousness or coma in the company of friends or by himself, but presents with a psychotic-like picture of confusion, incoherent speech, paranoia, assaultiveness, restlessness, autism, preoccupation, mutism, auditory hallucinations, and grandiosity. Entering the health care system at this point, without a ready history of drug intoxication, the patient easily and frequently is misdiagnosed as schizophrenic. This diagnosis does not complicate his or her immediate treatment, which is much the same as for acute schizophrenia, but labels him with a diagnosis which may be quite inappropriate and misleading for future medical contacts.

b. PCP Psychosis—A Psychiatric Emergency—Phencyclidine psychosis constitutes, in this initial phase, a psychiatric emergency. The patients are dangerous to themselves, because of depression and suicidal impulses, and dangerous to others, because of paranoia and strong tendencies toward violence. For patients fitting this description, psychiatric inpatient treatment is indicated. The immediate goals of treatment for this acute PCP-induced psychosis (Luisada and Brown 1976) are:

1. prevention of injury to the patient or others
2. assurance of continuing treatment
3. reduction of stimuli
4. amelioration of psychosis
5. reduction of agitation

c. Treatment of PCP Psychosis—Prior to instituting any phenothiazine treatment of a psychotic patient with a history of PCP use, it is essential to determine whether the patient is still intoxicated. The patient should be examined for the presence of nystagmus and ataxia, which, if present, indicate intoxication. The pupils should also be examined to guard against anticholinergic drug intoxication.

If there is no possibility of continuing intoxication by a cholinergic drug, chlorpromazine may be given, starting with 400 mg/day orally in divided doses (after a test dose of 50 mg) and increased as necessary by 200-600 mg/day. On the average, a daily dose of 1600 mg/day will be reached by the end of the initial phase of treatment. The response of PCP psychosis to this aggressive treatment is characteristically slow, distinguishing it from paranoid schizophrenia, which usually responds much more rapidly. Some clinicians prefer to use Haldol® (haloperidol), 5 mg IM given hourly if needed, to avoid the possibility of potentiation of PCP anticholinergic effects by a phenothiazine.

As the psychotic symptomatology abates, there are sometimes unpredictable reexacerbations. Patients often will wish to sign out against medical advice, with encouragement from others to do so, only to experience unexpected suicidal or homicidal impulses following their discharge (the patient still may not realize, because of drug-induced amnesia, that PCP was responsible for his hospitalization). When feasible, group and milieu psychotherapy can help.

Outpatient follow-up treatment is aimed at keeping the patient away from resuming drug use, a task which is not always easy despite the dysphoric experience which led to hospitalization. Luisada and Brown (1976) make the provocative observation that "about one-fourth of the patients originally treated for phencyclidine psychoses return about a year later with schizophrenic ones in the absence of drug use . . . These later episodes have lacked the characteristic violence of the phencyclidine-induced ones, and they have been much more quickly responsive to antipsychotic drugs." Thus, some of the users who became psychotic under PCP may have been "at risk" for schizophrenia, which later reappeared without the precipitating drug. (Some may, in fact, have been identifiably schizophrenic prior to using the drug.) In addition, significant numbers of users experience profound depression and must be followed carefully to monitor their suicidal potential (Smith *et al.* 1978).

PHENCYCLIDINE—THE UNANSWERED QUESTIONS

Our present knowledge of phencyclidine as an abused drug is decidedly modest. Indeed, as was pointed out earlier in this account, even the basic data pertaining to the incidence and prevalence of its abuse are largely lacking. What information is available represents a minimal estimate of both the extent of PCP use and of possible adverse effects of such use. Since PCP is often misrepresented as some other drug, inquiring about its use is certain to represent an underestimate of such use, especially by the less chronic user. And, as we have seen, the individual who suffers from a schizophrenic-like reaction to the drug is likely to be diagnosed as an acute schizophrenic reaction rather than as a toxic drug reaction.

Our present knowledge of the personality characteristics of more chronic users and the role that phencyclidine plays in their lives is also limited. For example, there is some question whether or not the acute schizophreniform reaction sometimes encountered in users may not be indicative of a latent schizophrenia precipitated by the drug. Similarly, we know little of the frequency and circumstances under which PCP potentiates aggression, although there have been many individual clinical reports of highly destructive behavior related to drug use.

Although it is believed that PCP ultimately results in "burned out," dulled intellectual functioning after repeated use, this has yet to be verified objectively. Besides possible adverse effects on intellectual functioning, phencyclidine may have adverse effects on psychosocial functioning that remain to be delineated. Because it is widely used during adolescence, it is important to know its possible effects on the ability to learn and to acquire other necessary skills.

A paradox that has not yet been explained is the apparent increase in PCP use despite the poor street reputation of the drug. This increased popularity may reflect a change in attitude toward the drug as a result of increased use by smoking as opposed to oral ingestion or it may reflect simply the widespread availability of phencyclidine because of the ease with which it can be synthesized. Motivations for use remain poorly understood.

Lasting biological implications of use and especially of chronic use are largely unknown. These include possible reproductive effects of PCP use as well as possible alterations in neurophysiological functioning that may be related to prolonged use. While higher doses of this drug are unquestionably incapacitating, much less is known about the effects of lower doses which may be more typical of recreational use, especially when the drug is smoked. Thus the wide range of possible dose response relationships involving PCP need elucidation. Carefully designed experiments which investigate the relationships between dose, tissue, and fluid levels, and pharmacological and behavioral effects of PCP and its analogs should be conducted in man where medically and ethically feasible. Since PCP is also not uncommonly used in association with such other drugs as alcohol, marihuana, and the barbiturates, it is important to know the effects of such likely combinations. For example, doses of alcohol or PCP which are not seriously toxic alone may become so when both drugs are taken concomitantly.

Similarities and differences between PCP and other abused substances can be evaluated in animals with sensitive behavioral techniques. Behavioral and drug-specific variables which contribute to the initiation and maintenance of PCP self-administration should be studied. The effects of PCP on a wide range of behaviors (e.g., aggression, punishment-induced conflict, cognitive function and sensory discrimination) remain to be investigated. The long-term behavioral-toxicological consequences of phencyclidine administration should be examined in these contexts.

Unfortunately, phencyclidine has about 30 analogs—drugs with slightly different chemical structures which nevertheless have similar effects. Little is known about the similarities or possible differences in effects between these compounds and PCP.

Recent attention to the detection and quantification of phencyclidine in biologic samples has provided valuable techniques, which are based on standard and existing methods. Additional evidence concerning their clinical validity is needed. There is considerable need for detailed information on the metabolism, disposition, and pharmacokinetics of phencyclidine and its analogs in acute and chronic situations.

Finally, it is important that we develop effective prevention strategies to discourage PCP use. This will necessitate understanding the reasons for its use and the most effective ways to reach and dissuade the user. These goals become particularly important for a drug, or family of drugs, whose street availability cannot be diminished substantially by means presently known.

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LETTER TO THE JOURNAL

Cancer Incidence and Estimated Mortality

November 20, 1978

Dear Dr. Krosnick:

Please read the article on cancer in the April 1978 issue of the *Bulletin of the American College of Surgeons.* There is much truth there—much more truth there than in all those articles in the October 1978 issue of *The Journal.* After directing two cancer clinics in New Jersey for about 35 years, I am surprised that the press and public accepts unproved statements.

At the recent directors' meeting of the American Cancer Society not one scientist could put an honest percentage on carcinogenic cancer.

Please examine the following data* as to where New Jersey stands in respect to other states of the union. These are recent figures. Note also the estimates of various states are close together. It is difficult to explain the high rates in Maine and some other states in chemicals.

(signed) William O. Wuester, M.D.

*Quoted from "Cancer Facts and Figures"—American Cancer Society

CANCER DEATHS – 1979

Estimated Cancer Deaths far All Sites plus Majar Sites, by State – 1979

State	All Sites		Majar Sites								
	Number af Deaths	Death Rate per 100,000 Papulatian	Female Breast	Calan-Rectum	Lung	Oral	Uterus	Prastate	Stamach	Pancreas	Leukemia
Alabama	6,300	168	450	650	1,700	125	250	375	200	375	200
Alaska	250	57	15	30	75	5	10	10	10	10	10
Arizana	3,700	152	250	375	1,000	100	50	200	125	150	125
Arkansas	4,300	195	250	450	1,200	100	100	275	150	250	200
Califarnia	37,900	169	3,300	4,400	9,500	850	1,000	1,800	1,500	1,800	1,400
Calarada	3,300	121	300	450	750	70	70	225	125	175	150
Cannecticut	6,200	198	550	900	1,400	175	125	275	250	325	250
Delaware	1,100	186	70	150	300	25	20	50	30	50	30
Dist. af Calumbia	1,600	238	175	200	350	75	60	100	60	80	40
Flarida	22,300	250	1,400	2,900	5,900	450	400	1,300	800	950	500
Geargia	7,400	143	600	750	2,100	200	275	400	225	350	275
Hawaii	1,100	118	60	100	250	30	20	40	90	60	50
Idaha	1,300	145	90	150	300	25	25	90	40	70	70
Illinais	20,900	185	2,000	3,000	5,100	500	700	1,100	750	1,100	900
Indiana	9,200	171	800	1,300	2,400	200	325	475	250	500	350
Iawa	5,600	194	500	900	1,300	100	125	350	150	250	275
Kansas	4,200	178	400	550	1,000	100	125	275	125	225	200
Kentucky	6,400	182	450	800	1,800	175	200	325	150	325	275
Lauisiana	6,600	165	475	700	1,800	175	200	375	250	300	250
Maine	2,200	198	175	325	550	50	60	125	90	100	80
Maryland	7,500	179	650	950	2,000	200	200	375	225	325	200
Massachusetts	11,800	203	1,200	1,800	2,600	325	300	600	500	600	400
Michigan	15,200	167	1,400	2,000	3,700	325	425	800	475	700	550
Minnesata	6,600	164	650	950	1,300	150	125	475	250	375	275
Mississippi	3,900	160	275	425	1,100	90	125	300	125	225	200
Missauri	9,400	199	800	1,300	2,500	200	275	550	275	475	400
Mantana	1,300	167	100	175	300	30	30	75	50	80	60
Nebraska	2,800	177	250	450	600	60	70	200	125	175	150
Nevada	950	141	60	100	300	25	20	30	10	60	30
New Hampshire	1,700	193	150	275	400	40	50	90	40	80	70
New Jersey	15,200	206	1,400	2,400	3,600	350	400	700	650	800	500
New Mexica	1,500	121	100	175	350	25	30	70	50	70	50
New Yark	37,000	208	4,000	5,800	8,200	850	1,000	1,700	1,600	2,100	1,400
Narth Caralina	8,800	156	650	850	2,100	225	300	500	250	425	350
Narth Dakata	1,100	166	90	150	200	20	25	80	60	80	50
Ohia	19,700	184	1,800	2,800	5,200	425	650	1,000	650	900	750
Oklahama	5,300	184	375	600	1,500	100	125	325	150	275	200
Oregan	4,200	171	325	500	1,200	100	100	225	125	200	200
Pennsylvania	24,500	208	2,300	3,700	5,700	500	750	1,200	850	1,200	950
Rhade Island	2,200	237	200	350	475	60	50	100	100	90	60
Sauth Caralina	4,300	146	325	400	1,200	100	150	275	125	250	150
Sauth Dakata	1,300	187	80	200	250	20	40	100	50	90	80
Tennessee	7,700	175	600	800	2,100	200	225	425	225	400	275
Texas	20,000	151	1,500	2,100	5,400	425	600	1,000	700	1,100	900
Utah	1,300	98	100	150	225	20	30	90	50	50	60
Vermant	850	172	70	150	225	20	30	60	30	50	40
Virginia	8,300	157	650	900	2,200	175	250	400	250	425	275
Washingtan	6,500	174	500	800	1,600	150	150	375	200	325	275
West Virginia	3,600	190	250	400	950	75	125	200	100	200	125
Wiscansin	8,100	172	800	1,200	1,600	175	200	475	325	400	325
Wyaming	550	129	40	70	150	10	10	40	15	30	20
United States	395,000	180	34,000	52,000	98,000	9,000	11,000	21,000	14,000	20,000	15,000
Puerta Rica	3,000	102	125	175	250	175	200	175	425	100	125

NEW CANCER CASES – 1979

Estimated New Concer Coses for All Sites Plus Major Sites, by State – 1979

State	All Sites*	Major Sites								
	Number of Cases	Female Breast	Colon-Rectum	Lung	Oral	Uterus (Invasive)	Prostate	Stomach	Pancreas	Leukemia
Alabama	13,000	1,500	1,500	2,000	350	1,000	1,000	300	400	350
Alaska	500	50	50	75	15	30	20	15	10	20
Arizona	7,600	1,000	900	1,100	175	600	600	150	200	200
Arkansas	8,300	900	1,000	1,400	200	550	800	200	250	300
California	73,000	11,100	9,700	11,000	2,400	4,700	5,800	2,300	2,300	2,100
Colorado	6,500	1,000	800	750	175	400	550	150	200	200
Connecticut	12,000	2,000	1,900	1,700	450	650	950	400	400	350
Delaware	2,100	250	300	300	70	150	150	50	50	40
Dist. of Columbia	3,100	450	400	400	200	250	250	90	100	60
Florida	42,000	5,300	6,300	6,800	1,300	2,700	4,000	1,000	1,200	1,000
Georgia	15,000	1,700	1,700	2,300	500	1,200	1,300	400	400	450
Hawaii	2,100	150	250	300	80	125	100	150	60	70
Idaho	2,400	300	300	300	50	150	250	70	80	100
Illinois	40,000	5,900	6,200	5,900	1,300	2,700	3,100	1,400	1,200	1,200
Indiana	18,000	2,400	2,700	2,600	475	1,600	1,400	400	500	500
Iowa	11,000	1,400	1,900	1,500	300	700	1,100	300	350	400
Kansas	8,100	1,200	1,100	1,200	250	650	800	150	250	300
Kentucky	12,000	1,400	1,700	2,100	425	950	1,100	250	350	400
Louisiana	13,000	1,500	1,500	2,100	475	850	1,200	400	400	350
Maine	4,100	600	700	600	125	300	400	150	150	100
Maryland	15,000	2,200	2,100	2,300	525	1,100	1,200	350	400	350
Massachusetts	23,000	3,400	3,800	3,000	875	1,400	1,800	850	650	600
Michigan	30,000	4,300	4,200	4,300	950	2,200	2,400	850	850	800
Minnesota	13,000	1,800	2,000	1,400	375	800	1,400	500	450	450
Mississippi	7,700	800	900	1,200	200	600	900	250	250	300
Missouri	19,000	2,300	2,900	2,800	525	1,400	1,700	450	550	550
Montana	2,300	300	350	300	70	150	250	80	80	80
Nebraska	5,300	700	900	650	150	350	500	150	200	225
Nevada	2,100	200	200	350	50	100	100	15	60	40
New Hampshire	3,200	400	550	450	100	225	250	70	90	100
New Jersey	29,000	4,500	4,900	4,200	900	2,100	2,300	1,100	900	750
New Mexico	3,000	300	350	350	60	200	250	80	90	70
New York	70,000	11,100	12,000	9,500	2,400	4,800	5,100	2,800	2,300	2,000
North Carolina	17,000	2,400	2,100	2,400	525	1,300	1,700	350	500	500
North Dakota	2,000	250	300	200	50	100	300	100	80	70
Ohio	38,000	5,400	6,000	5,900	1,200	2,800	3,000	1,200	1,100	1,100
Oklahoma	10,000	1,200	1,400	1,700	275	650	900	250	300	300
Oregon	8,100	1,100	1,000	1,300	200	500	700	200	250	300
Pennsylvania	48,000	6,800	8,700	6,600	1,500	3,200	3,600	1,600	1,300	1,300
Rhode Island	4,100	600	750	500	200	300	300	150	100	80
South Carolina	8,200	1,000	950	1,300	275	700	800	150	250	225
South Dakota	2,400	300	400	250	50	150	300	80	90	100
Tennessee	15,000	1,900	1,900	2,400	475	1,100	1,200	300	450	450
Texas	39,000	4,700	4,600	6,300	1,100	2,700	3,300	1,100	1,200	1,200
Utah	2,500	350	350	250	50	200	300	80	80	80
Vermont	1,600	200	300	225	60	150	200	50	50	60
Virginia	16,000	2,300	2,000	2,500	525	1,100	1,200	350	450	400
Washington	13,000	1,900	1,700	1,900	350	750	1,100	350	350	400
West Virginia	6,700	800	900	1,100	200	600	600	200	200	150
Wisconsin	16,000	2,300	2,500	1,800	450	950	1,400	600	500	450
Wyoming	1,000	100	100	150	20	70	80	20	30	30
United States	765,000	106,000	112,000	112,000	24,000	53,000	64,000	23,000	23,000	22,000
Puerto Rico	6,000	350	275	300	425	700	300	500	100	125

*Does not include carcinoma in situ or non-melanoma skin cancers.

These estimates are offered as a rough guide and should not be regarded as definitive. They are calculated according to the distribution of estimated 1979 cancer deaths by state. Especially note that year-to-year changes may only represent improvements in the basic data.

the physician and sportsmedicine

COLLEGE OF MEDICINE AND DENTISTRY
OF NEW JERSEY

Baseboll Injuries—Prevention ond Manogement
A Continuing Educotion Conference
Morch 29, 1979, 8:00 AM—5:30 PM

Presented by The Physicion ond Sportsmedicine
ond CMDNJ—New Jersey Medical School
ot the College's Medicol Science Building,
New Jersey Medical School, Newark, NJ

Speokers include the teom physicians for
The New York Yonkees, Boston Red Sox, and
Philodelphio Phillies, the President of Little
League Boseboll ond other noted Sports
Medicine speciolists.

Opportunities for CME credits will be available.

For further informotion contact
CMDNJ—Deportment of Continuing Education
Box 101, University Heights
Piscotowoy, New Jersey 08854
Call 201/463-4707

WEEKLY CARDIOLOGY CONFERENCES

(Approved for 2 hours Cot. I Credit for C.M.E.)

Objective of Program: Updote of diognostic methods in cordiology; indicotions ond results of cordioc surgery.

PROGRAM CHAIRMAN: Horry Goldberg, M.D., Professor of Medicine
Temple University School of Medicine
Chief of Cordiology, Deboroh Heort ond Lung Center

With speciolists on Deboroh's stoff presenting Echocordiogroms, ECG's, Treodmill testing, Vector-Cardiogroms, Nucleor Scons, Coronory Cines ond Ventriculogroms, Cordioc Coth. Doto, Angiograms ond Surgicol Methods.

Guest Lecturers: A distinguished guest lecturer will present speciol topics on Cordiology once o month.

THURSDAYS (Every Week) 2:15 P.M.—4:00 P.M. (September 7, 1978 to June 28, 1979)

LOCATION: DEBORAH HEART AND LUNG CENTER,
BROWNS MILLS
(1st. Floor Conf. Room)

THE DEPARTMENT OF MEDICINE and
THE PAGE AND WILLIAM BLACK
POST-GRADUATE SCHOOL OF MEDICINE
of the MOUNT SINAI SCHOOL OF MEDICINE (CUNY)

Announce a Postgraduate Course

New Approaches to Old Problems in Internal Medicine

Under the Direction of
RICHARD GORLIN, M.D. HARRIET S. GILBERT, M.D.
and FACULTY of the MOUNT SINAI SCHOOL OF MEDICINE

May 15-18, 1979

Tuesday-Thursday, 9:00 AM to 5:00 PM; Friday, 9:00 AM to 4:00 PM, (4 Sessions)

The proctice of Internol Medicine presents o continuing chollenge to integrote new concepts of diognosis and therapy into the context of potient monogement. This four-doy multidisciplinary course in Internol Medicine is designed for the procticing physician ond will cut ocross subspeciolties to address commonly encountered clinical situotions by means of concise lectures, ponel discussions ond workshops. Each session will be problem-oriented ond bosed on illustrotive case moteriol. Opportunity will be provided for registronts to clorify questions orising from their own clinicol experience through question-ond-onswer periods ond informol discussions with foculty.

One afternoon will be devoted to porticipotion in smoll subspeciolty workshops or may be spent with members of the faculty who will be available by odvonce orrongement to discuss topics of particulor interest to the registrants. This session will be followed by ottendance ot Medicol Grand Rounds.

A syllobus summorizing the questions to be oddressed by each speaker, pertinent clinicol moteriol, ond references will be distributed.

FEE: $300.00 (including Luncheon ond Cocktoil Porty)

This course will be given at the Mount Sinai Medical Center, New York, New York

APPLY TO: Director, The Poge ond Williom Block Post-Groduote School of Medicine, Mount Sinai School of Medicine, One Gustove L. Levy Ploce, New York, New York 10029.
Tel: (212) 650-6737.

CME CALENDAR

This listing is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, the Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the College of Medicine and Dentistry of New Jersey. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

ANESTHESIOLOGY

March

24-25 **20th Annual Postgraduate Seminar**
9 a.m.-5 p.m., Sat.: 9 a.m.- 1 p.m., Sun.
—Hyatt House, Cherry Hill
(New Jersey State Society of Anesthesiologists and AMNJ)

MEDICINE (includes Family, Internal, and General Medicine and Dermatology)

March

1 **Cardiology Conferences**
8 2:15-4:15 p.m.—Deborah Heart and
15 Lung Center, Browns Mills
22 *(Deborah Heart and Lung Center and*
29 *AMNJ)*

2 **Lymphomas**
9 **Carcinoma of the Colon**
14 **CA of the Cervix**
16 **CA of the Urinary Tract**
23 **Chemotherapy Review**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

6 **Hypertension in Cardio-Vascular Renal Disease**

13 **Use of Psychtrophic Drugs in Cardiac Patients**
8-9 a.m.—Greater Paterson General Hospital
(Greater Paterson General Hospital and AMNJ)

6 **Controversies in Cardiology**
9-10 a.m.—Holy Name Hospital, Teaneck
(Holy Name Hospital and AMNJ)

7 **Dinner Meeting**
6:30-9:30 p.m.—Holiday Inn, East Orange
(AMNJ Endocrinology Section)

7 **Medical Grand Rounds**
11:30 a.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

7 **Joint Conference—NJ Thoracic Society and ACCP**
9 a.m.-4 p.m.—Coachman Inn, Cranford
(NJ Thoracic Society, NJ Chapter, American College of Chest Physicians, and AMNJ)

7 **Human Immunologic Response in Lymphocyte Function**
14 **Nuclear Cardiology**
21 **Infectious Diseases**
9-11 a.m.—Riverview Hospital, Red Bank
(Riverview Hospital and AMNJ)

7 **Gall Bladder Disease**
14 **Diabetic Retinopathy and Retinal Neo-Vascularization**
28 **Renal Failure**
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

7 **Advances in Medicine**
14 9:30-11 a.m.—Bergen Pines County
21 Hospital, Paramus
28 *(Bergen Pines County Hospital and AMNJ)*

14 **Diabetes**
21 **Orthotics and Prosthetics**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ and AAFP)

7 **Endocrine Conferences**
14 3:30-5 p.m.—Rotates between Newark
21 Beth Israel Medical Center, Martland
28 Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

7 **Vasodilator Therapy in Heart Disease**
14 **Current Problems in Transplantation**
21 **Common Gastrointestinal Disorders Among Children**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

8 **Virology**
15 4-6 p.m.—Institute for Medical
22 Research, Copewood St., Camden
29 *(Institute for Medical Research and AMNJ)*

10 **Vascular Injuries**
1-5 p.m.—Newark Beth Israel Medical Center
(N.J. Chapter, American Trauma Society, Vascular Society of N.J. and AMNJ)

13 **Immunology**
8-10 p.m.—Schering Corporation, Kenilworth
(NJ Dermatological Society and AMNJ)

13 **Acute Renal Failure**
20 **Chronic Renal Failure**
27 **Fluid and Electrolyte Imbalance**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

13 **Nutrition and Aging**
8 p.m.—Paul Kimball Hospital, Lakewood
(Paul Kimball Hospital and AMNJ)

14 **Medical Lecture Series**
21 1-3 p.m.—Christ Hospital, Jersey City
28 *(Christ Hospital and AMNJ)*

15 **Treatment of Bronchospasm**
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

15 **Regional Chest Conferences**
7:30-9:30 p.m.—Overlook Hospital,, Summit
(N.J. Thoracic Society and AMNJ)

20 **Gastroenterology—Proper Use of Endoscopy**
8:30-9:30 p.m.—Omar's Restaurant, Saddle River Rd., Fair Lawn
(Fair Lawn Memorial Hospital and AMNJ)

20 **Nuclear Medicine**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

27 **Head and Neck Cancer**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

28 **Cardiac Grand Rounds**
3-4:30 p.m.—NJ Medical School, Newark
(American Heart Association, NJ Affiliate and AMNJ)

28 **Current Concepts in Cardiac Arrhythmias**
12 noon—Town and Campus, West Orange
(Essex County Heart Association, Chapter of NJ Heart Association)

April

4 **Congenital Heart in Adults**
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

4 **Medical Grand Rounds**
11:30 a.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

4 **Lung Disorders and Blood Gases**
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

4 **Nonmalignant Disorders of the Bowel**
11 **Fever of Unknown Origin**
18 **Contact Dermatitis 1979**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

4 **Dinner Meeting**
6:30-9:30 p.m.—Holiday Inn, East Orange
(AMNJ Endocrinology Section)

4 **Nutrition-Elemental Diets**

18 **Medieal Leeture Series**
25 1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

4 **Endocrine Conferences**
11 3:30-5 p.m.—Rotates between Newark
18 Beth Israel Medical Center, Martland
25 Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

4 **Advanees in Medicine**
11 9:30-11 a.m.—Bergen Pines County
18 Hospital, Paramus
25 *(Bergen Pines County Hospital and AMNJ)*

5 **Cardiology Conferences**
12 2:15-4:15 p.m.—Deborah Heart and Lung
19 Center, Browns Mills
26 *(Deborah Heart and Lung Center and AMNJ)*

6 **Arthritis**
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

6 **Acute Respiratory Failure**
13 **Bronchial Asthma Update**
20 **COPD and Rehabilitation**
27 **O_2 Therapy**
11:45 a.m.-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Hospital and AMNJ)

10 **Sudden Death in Young Athletes**
12:45-2 p.m.—Mercer Medical Center, Trenton
(American Heart Association, Mercer County Chapter)

10 **Endoerinology and Metabolism Testing**
9 p.m.—West Hudson Hospital, Kearny
(West Hudson Hospital and AMNJ)

10 **Immunology**
8-10 p.m,—Schering Corporation, Kenilworth
(NJ Dermatological Society and AMNJ)

12 **Case Presentations and Guest Speakers**
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

12 **Virology**
19 4-6 p.m.—Institute for Medical Research,
26 Copewood St., Camden
(Institute for Medical Research and AMNJ)

18 **Thromboembolic Disease**
11:30 a.m.-1 p.m.—VA Hospital, East Orange
(VA Hospital and AMNJ)

19 **Regional Chest Conferences**
7:30-9:30 p.m.—St. Joseph's Medical Center, Paterson
(NJ Thoracic Society and AMNJ)

19 **Juvenile Diabetes**
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

24 **Upper Gastrointestinal Bleeding**
8-9 a.m.—Greater Paterson General Hospital, Wayne
(Greater Paterson General Hospital and AMNJ)

24 **Lower Back Pain**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

25 **Cardiae Grand Rounds**
3-4:30 p.m.—NJ Medical School, Newark
(American Heart Association, NJ Affiliate, and AMNJ)

25 **William P. Burpeau Award Dinner**
6:30 p.m.—The Manor, West Orange
(Urology Section of AMNJ)

NEUROLOGY/PSYCHIATRY

Mareh

1 **Prediction of Response to Treatment with Antidepressants**
8 **Cultural Psyehiatry**
15 **Emotional Foundations of Happiness**
22 12 noon-1 p.m.—Carrier Foundation,
29 Belle Mead
(Carrier Foundation)

1 **Improving Psyehotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, South Orange
(Group for Advanced Psychiatric Study and AMNJ)

1 **Psychiatrie Lecture Series**
8 11 a.m.-12 noon—Greystone Park
15 Psychiatric Hospital
22 *(Greystone Park Psychiatric Hospital and*
29 *AMNJ)*

2 **Psychiatrie Lecture Series**
9 1:30-5 p.m.—Trenton Psychiatric
16 Hospital
23 *(Trenton Psychiatric Hospital and*
30 *AMNJ)*

5 **An Adolescent Girl in a Lolie à deux**
8-10 p.m.—111 Ridgewood Ave., Glen Ridge
(Essex Psychiatric Seminar and AMNJ)

5 **Neuroscience Conferenees**
12 11:30 a.m.-12:30 p.m.—Bergen Pines
19 County Hospital, Paramus
26 *Bergen Pines County Hospital and AMNJ)*

6 **Introduetion to Family Therapy**
13 7:30-9:30 a.m.—Trenton Psychiatric Hospital
(Trenton Psychiatric Hospital and AMNJ)

6 **Seminar on Law and Psychiatry**
13 3:30-5:30 p.m.—Rutgers Law School,
20 Newark
27 *(Rutgers University Law School and AMNJ)*

7 **Psyehological Sequel of Steroid Hormone Exposure**
21 **Mental Health Revolution**
1:30-3 p.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

7 **Forensie Psyehiatry—Coneepts and Areas of Coneern**
14 **Forensic Psyehiatry—Reeent Trends**
21 **Phohie and OBS, Compulsive Neurosis**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

7 **Course for Psyehiatrists—Semester II**
14 8-10 p.m.—Hackensack Hospital
21 *(NJ Psychoanalytic Society and AMNJ)*
28

23 **Joint Meeting with New Jersey Psychiatric Association**
8:30-10:30 p.m.—Mayfair Farms, West Orange
(New Jersey Psychoanalytic Society and AMNJ)

23 **Young Freud & Friends—Joint Meeting**
8-10 p.m.—Town & Campus, West Orange
(NJ Psychoanalytic Society, NJ Psychiatric Association and AMNJ)

28 **Neuropathology Conferenees**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

28 **Psychopathology of Sexual Dysfunction**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ, and AAFP)

30 **Handling the Patient with Cancer—Psychiatric Considerations**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

April

2 **Voyeurism and Sexual Deviation**
8-10 p.m.—60 Melrose Place, Montclair
(Essex Psychiatric Seminar and AMNJ)

2 **Neuroseience Conferenees**
9 11:30 a.m.-12:30 p.m.—Bergen Pines
16 County Hospital, Paramus
23 *(Bergen Pines County Hospital and*
30 *AMNJ)*

3 **Seminar on Law and Psyehiatry**
10 3:30-5:30 p.m.—Rutgers Law School,
17 Newark
24 *(Rutgers University Law School and AMNJ)*

4 **Infant Day Care**
7:30-9:30 p.m.—Marriott Motor Inn, Saddle Brook
(NJ Psychiatric Association, NJ Council of Child and Adolescent Psychiatry and AMNJ)

4 **Delayed Grief Reaetions**
18 **Hyperaetive Children**
1:30-3 p.m.—NJ Medical School, Newark
(NJ Medical School, Dept. of Psychiatry)

4 **Violence-Evaluation/Coneepts**
18 **Psychologieal Testing**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

4 **Course for Psychiatrists—Semester II**
11 8-10 p.m.—Hackensack Hospital
18 *(NJ Psychoanalytic Society and AMNJ)*

5 **Psyehotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, South Orange
(Group for Advanced Psychiatric Study and AMNJ)

5 **Psychiatrie Leeture Series**
12 11 a.m.-12 noon—Greystone Park
19 Psychiatric Hospital
26 *(Greystone Park Psychiatric Hospital and AMNJ)*

5 **Schizophrenic Syndrome**
12 **Psyehoneuroendocrinology Psychiatry**
19 **Cerebral Dysrhythmia and Schizophrenia**
26 12 noon-1 p.m.—Carrier Foundation, Belle Mead
(Carrier Foundation)

6 **Psychiatric Leeture Series**
20 1:30-5 p.m.—Trenton Psychiatric
27 Hospital
(Trenton Psychiatric Hospital and AMNJ)

7 **Pediatrie Neurology**
Morristown Memorial Hospital
(Morristown Memorial Hospital and AMNJ)

10 **Cerebral Vaseular Disease**

17 Headache
24 Seizure Disorders
11 a.m.—12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

11 Psycho-Sexual Dysfunctions in Clinical Practice
8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

11 Sleep Disorders
8:30-10:30 p.m.—Guido's Restaurant, Hackensack
(North Jersey Psychiatric Society and AMNJ)

11 Schizophrenia in a State Hospital
1:15-2:45 p.m.—Marlboro Psychiatric Hospital
(Marlboro Psychiatric Hospital and AMNJ)

11 Depression and Anxiety
1:30-2:30 p.m.—John E. Runnells Hospital of Berkeley Heights
(John E. Runnells Hospital and AMNJ)

OBSTETRICS/GYNECOLOGY

March

1 Grand Rounds and Case Presentations
8 4-5 p.m.—Martland Hospital, Newark
15 *(CMDNJ and AMNJ)*
22
29

2 Annual Edward G. Waters Gynecologic
4 Conference
Begins 8:30 a.m.—Playboy Club, Great Gorge
(CMDNJ and AMNJ)

7 Lectures in Obstetrics and Gynecology
8-10 p.m.—Location varies
(CMDNJ and AMNJ)

7 Grand Rounds and Case Presentations
14 2-4 p.m.—Rotating between Martland
21 Hospital, Newark Beth Israel,
28 St. Michael's, St. Joseph's Hospital and Jersey City Medical Centers
(CMDNJ and AMNJ)

April

4 Lectures in Obstetrics/Gynecology
8-10 p.m.—Location varies
(CMDNJ and AMNJ)

4 Grand Rounds and Case Presentations
11 2-4 p.m.—Rotating between Martland
18 Medical Center, Newark Beth Israel,
25 St. Michael's, St. Joseph's Hospitals and Jersey City Medical Center
(CMDNJ and AMNJ)

5 Grand Rounds and Case Presentations
12 4-5 p.m.—Martland Hospital,
19 Newark
26 *(CMDNJ and AMNJ)*

11 Infertility
11:45 a.m.-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Hospital and AMNJ)

PATHOLOGY/CLINICAL PATHOLOGY

March

7 Clinical Pathology Grand Rounds
14 12 noon-1 p.m.—NJ Medical School,
21 Newark
28 *(CMDNJ and AMNJ)*

April

4 Clinical Pathology Grand Rounds
11 12 noon-1 p.m.—NJ Medical School, Newark
18 *(CMDNJ and AMNJ)*
25

25 Neuropathology Conferences
8-9:15 a.m.—NJ Medical School
(CMDNJ and AMNJ)

PEDIATRICS

March

2 Advances in Pediatrics
9 9:30-10:30 a.m.—NJ Medical School,
16 Newark
23 *(CMDNJ and AMNJ)*
30

4 Infant Day Care
7:30-9:30 p.m.—Marriott Motor Inn, Saddle Brook
(NJ Psychiatric Association, NJ Council of Child and Adolescent Psychiatry, and AMNJ)

6 Pediatric Surgery
13 8-9 a.m.—Garden State Community Hospital
(Garden State Community Hospital and AMNJ)

April

6 Advances in Pediatrics
20 9:30-10:30 a.m.—NJ Medical School, Newark
27 *(CMDNJ and AMNJ)*

7 Pediatric Neurology
Morristown Memorial Hospital
(Morristown Memorial and AMNJ)

18 Pediatric Dentistry
8 a.m.-3:30 p.m.—VA Hospital, East Orange
(AMNJ)

19 Juvenile Diabetes
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

RADIOLOGY

March

8 Case Presentations and Guest Speakers
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

15 Combined Radiology and Orthopaedic Meeting
7:15-10:15—Hospital Center at Orange
(Diagnostic Radiology Section for Northern NJ and AMNJ)

21 Radiotherapy Section Dinner Meeting
6:30 p.m.-To be announced
(AMNJ)

April

19 Ultrasound
7:15-10:15 p.m.—Hospital Center at Orange
(Diagnostic Radiology Section for Northern NJ and AMNJ)

GENERAL SURGERY

March

5 Lecture Series in Surgery
12 4:30-5:30 p.m.—NJ Medical School,
19 Newark
26 *(CMDNJ and AMNJ)*

6 Weekly Tumor Conferences
13 11 a.m.-12 noon—Morristown
20 Memorial Hospital
27 *(Morristown Memorial Hospital and AMNJ)*

9 Carcinoma of the Colon
14 CA of the Cervix
16 CA of the Urinary Tract
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

10 Vascular Injuries
1-5 p.m.—Newark Beth Israel Medical Center
(NJ Chapter, American Trauma Society, Vascular Society of NJ and AMNJ)

14 Current Problems in Transplantation
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ, and AAFP)

20 Surgical Management—Ulcerative Colitis
7-8 p.m.—Irvington General Hospital
(Irvington General Hospital and AMNJ)

27 Head and Neck Cancer
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

28 Patient Evaluation for Surgical Procedures
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ, and AAFP)

April

2 Lecture Series in Surgery
9 4:30-5:30 p.m.—NJ Medical School,
16 Newark
23 *(CMDNJ and AMNJ)*
30

3 Weekly Tumor Conferences
10 11 a.m.-12 noon—Morristown
17 Memorial Hospital
24 *(Morristown Memorial Hospital and AMNJ)*

17 Colon and Rectal Cancer
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

21 Cancer Seminar
8:30 a.m.-12:30 p.m.—Ramada Inn, New Brunswick
(Philippine-American Medical Society of NJ and AMNJ)

25 Colorectal Polyps and Cancers
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

SURGICAL SPECIALTIES (includes ENT, neurosurgery, ophthalmology, orthopedics, plastics, and vascular surgery)

March

1 Neurosurgical Case Presentations
8 4-5:30 p.m.—NJ Medical School,
15 Newark
22 *(CMDNJ and AMNJ)*
29

2 Diagnostic and Therapeutic Problems in
9 Orthopedics
23 7:30-9 a.m.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital)

6 Pediatric Surgery
13 8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

6 **Controversial Topics in Esophageal Surgery**
8-10 p.m.—Hilton Hotel, Meadowlands
(N.J. Society of Thoracic Surgeons and AMNJ)

7 **Orthopedic Problems: Bone Tumors**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

10 **Vascular Injuries**
1-5 p.m.—Newark Beth Israel Medical Center
(N.J. Chapter, American Trauma Society, Vascular Society of N.J. and AMNJ)

14 **Current Problems in Transplantation**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ, and AAFP)

14 **Endometrial Carcinoma**
8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

15 **Combined Radiology and Orthopaedic Meeting**
7:15-10:15 p.m.—Hospital Center at Orange
(Diagnostic Radiology Section for Northern NJ and AMNJ)

27 **Vascular Reconstructive Surgery**
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

30 **Fourth Annual Orthopaedic Symposium**
31 8 a.m.—Rutgers Medical School, Piscataway
(CMDNJ and AMNJ)

April

5 **Neurosurgical Case Presentations**
12 4-5:30 p.m.—NJ Medical School,
19 Newark
(CMDNJ and AMNJ)

6 **Orthopedics**
13 7:30-9 a.m.—Alexian Brothers Hospital,
27 Elizabeth
(Alexian Brothers Hospital)

24 **Lower Back Pain**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

24 **Hemi-Orthoplasty in Acute Femoral Neck Fractures**
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

MISCELLANEOUS

March

2 **Pharmacology, Clinical**
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

7 **Malpractice**
11:30 a.m.-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

7 **Clinical Pharmacology and Management**
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

7 **Doctors' Time and How to Manage it—**
8:30-11:30 a.m.
Medical Collection Management—
1-4:45 p.m.
Quality Inn, 283 Rt. 17 So., Hasbrouck Heights
(Bergen County Medical Society, AMA and AMNJ)

13 **Periodontal Signs and Symptoms**
6 p.m.—Ramada Inn, Rochelle Park
(AMNJ)

21 **Orthotics and Prosthetics**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital, AMNJ and AAFP)

April

3 **Malpractice**
8-9 p.m.—Burdette Tomlin Memorial Hospital, Cape May Court House
(Burdette Tomlin Memorial Hospital and AMNJ)

4 **Clinical Pharmacology**
11:30 a.m.-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

18 **Dental Symposium**
(All Day) VA Hospital, East Orange
VA Hospital and AMNJ)

FOR OCCUPATIONAL PHYSICIANS

A meeting on Employment of Disabled Citizens will be held on April 4, 1979, at Rutgers University Student Center in Newark. Agenda will include a workshop for physicians. Employment of the Handicapped under Section 503 of the Rehabilitation Act will be stressed.

FOR INFORMATION: Daniel J. O'Regan, M.D., Medical Director, New Jersey Division of Vocational Rehabilitation Services, Labor and Industry Building, John Fitch Plaza, Trenton, 08620, (609) 292-5987.

OBITUARIES

Dr. Robert Applestein

One of Mercer County's well-known practitioners, Robert Applestein, M.D., died suddenly on November 27, 1978 of an apparent heart attack. A native of Baltimore, born in 1904, and graduated from Jefferson Medical College, class of 1928, Dr. Applestein took graduate training in dermatology which he practiced exclusively since 1930 in Trenton. He was affiliated with the Mercer Medical Center there.

Dr. William H. Brown

One of Union County's senior members, William H. Brown, M.D., of Elizabeth, died at his home on December 11, 1978. A native of South Carolina, born in 1894, Dr. Brown earned his medical degree from Howard University, class of 1928, and came to New Jersey to practice family medicine in the Elizabeth area, where he remained until retirement in the early 1970s. He had been affiliated with Elizabeth General, Alexian Brothers, and St. Elizabeth Hospitals in Elizabeth and with Martland Medical Center in Newark.

Dr. Ralph K. Bush

One of Camden County's senior emeritus members, Ralph K. Bush, M.D., died on December 11, 1978, after a long illness. Born at the turn of the century, Dr. Bush was graduated from the University of Pennsylvania School of Medicine in 1923. He practiced family medicine in Merchantville and surrounding community until illness forced his retirement in 1976. He had been active in medical affairs and on the state level had been a member of MSNJ's Advisory Committee to the Women's Auxiliary. In 1973 Dr. Bush was recipient of the Medical Society's Golden Merit Award denoting fifty years of medical practice.

Dr. Leopold S. David

Leopold S. David, M.D., died on December 23, 1978 at his home in Cherry Hill. Born in 1906, and graduated from Hahnemann Medical College, class of 1931, Dr. David pursued graduate work in orthopedics and practiced industrial medicine for many years in Camden. During World War II Dr. David served in the medical department of the AUS. He was a Fellow of the American College of Legal Medicine.

Dr. Victor S. DiLeo

Victor S. DiLeo, M.D., a member of our Essex County component, died on October 17, 1978 after a long illness. Born in Port Chester, New York in 1909, Dr. DiLeo earned his medical degree from the University of Naples School of Medicine (Italy) in 1935 and returned to his native land to take graduate training in anesthesiology. He had been chief of that service at St. Mary's Hospital in Orange. Dr. DiLeo was a member of the American Society of Anesthesiologists and of the Academy of Medicine of New Jersey. During World War II he served for four years with the medical department of the Army of the United States.

Dr. James R. Irwin

Word just has been received of the death on August 28, 1973 of James R. Irwin, M.D., an emeritus member of our Essex County component, who formerly practiced in Belleville. A graduate of the University of Pennsylvania School of Medicine in 1919, Dr. Irwin pursued a career in surgery, earning Fellowship in the American College of Surgeons. He retired in 1955 and moved to California. His most recent address had been San Luis Obispo. Dr. Irwin was 86 years of age at the time of his death.

Dr. Harold F. Johnson

Word has been received of the death, from complications associated with pneumonia, on November 9, 1978, of Harold F. Johnson, M.D. Graduated from Columbia University College of Physicians and Surgeons in 1915, Dr. Johnson had practiced general medicine with special interest in pediatrics in the Plainfield area until retirement in 1971 because of illness. During World War I Dr. Johnson served with the army medical corps attached to the British Army. He was 90 years old at the time of his death.

Dr. Emanuel Katz

On October 8, 1978, Emanuel Katz, M.D., formerly a member of the Middlesex and Salem County Medical Societies, died of a heart ailment at his home. A native of New Jersey, born in 1915, Dr. Katz was graduated from the Royal Medical College in Scotland in 1947 and returned to New Jersey for internship in Trenton and New Brunswick and graduate training in neurology and psychiatry at the Veterans Administration Hospital in Lyons. He was in private practice in New Brunswick until 1970, during which time he was on the staffs at St. Peter's and Middlesex General Hospitals. At the time of his death he was affiliated with the Psychiatric Hospital in Marlboro.

Dr. A. T. Marchigiano

Word just has been received of the death on November 24, 1978 in Cheshire Hospital, Marlborough, New Hampshire of Anthony T. Marchigiano, M.D., a member of our Essex County component. Born in 1922 and graduated from Creighton University School of Medicine in 1947, Dr. Marchigiano returned to his native town of Newark to practice family medicine. He had been on the staff of the Presbyterian Hospital there.

Dr. J. Marshall Ney

Julian Marshall Ney, M.D., formerly of Newark and a member of our Essex County component, died on November 3, 1978 in Florida where he was living. A graduate of Emory University Medical School in 1924, Dr. Ney practiced ophthalmology and otolaryngology before retirement in 1975, when he moved to Maplewood. He had been affiliated with the Newark Eye and Ear Infirmary, Martland Medical Center in Newark, and Saint Barnabas Medical Center in Livingston. Dr. Ney was 78 years old at the time of his death.

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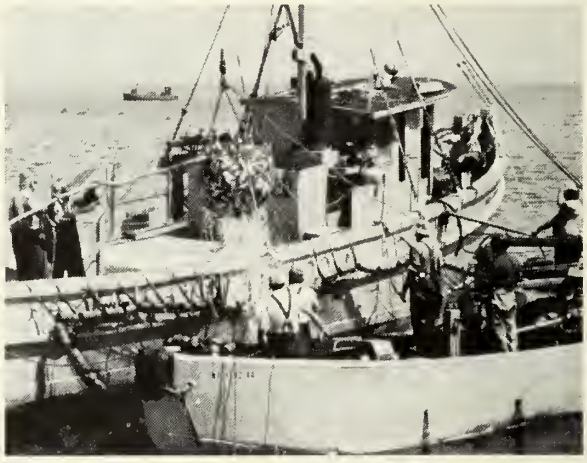
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For more information write to:

Dr. Robert Ellis, Executive Director
New Jersey Marine Science Consortium
101 College Road East, Princeton, N.J. 08540
609-452-8465

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GASTROENTEROLOGIST/INTERNIST—AOA, ABIM, GI Boards. 32, University trained, endoscopist on staff. Available immediately. Solo, group or partnership. Contact: H. Cohen, 8 Deerwood West, Apt. 44C, Irvine, Ca. 92714.

HEMATOLOGIST/ONCOLOGIST—Seeks association with group, partnership or hospital based practice. 31, certified ABIM and subspecialty board in hematology. Available July 1979. Write Box No. 109, c/o THE JOURNAL.

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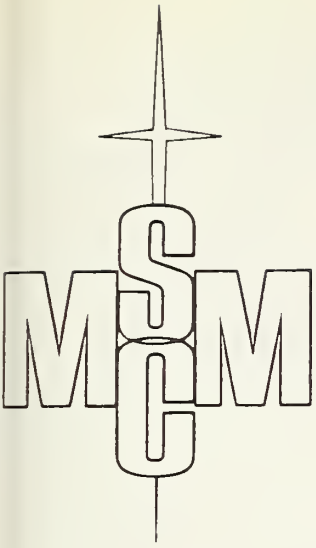
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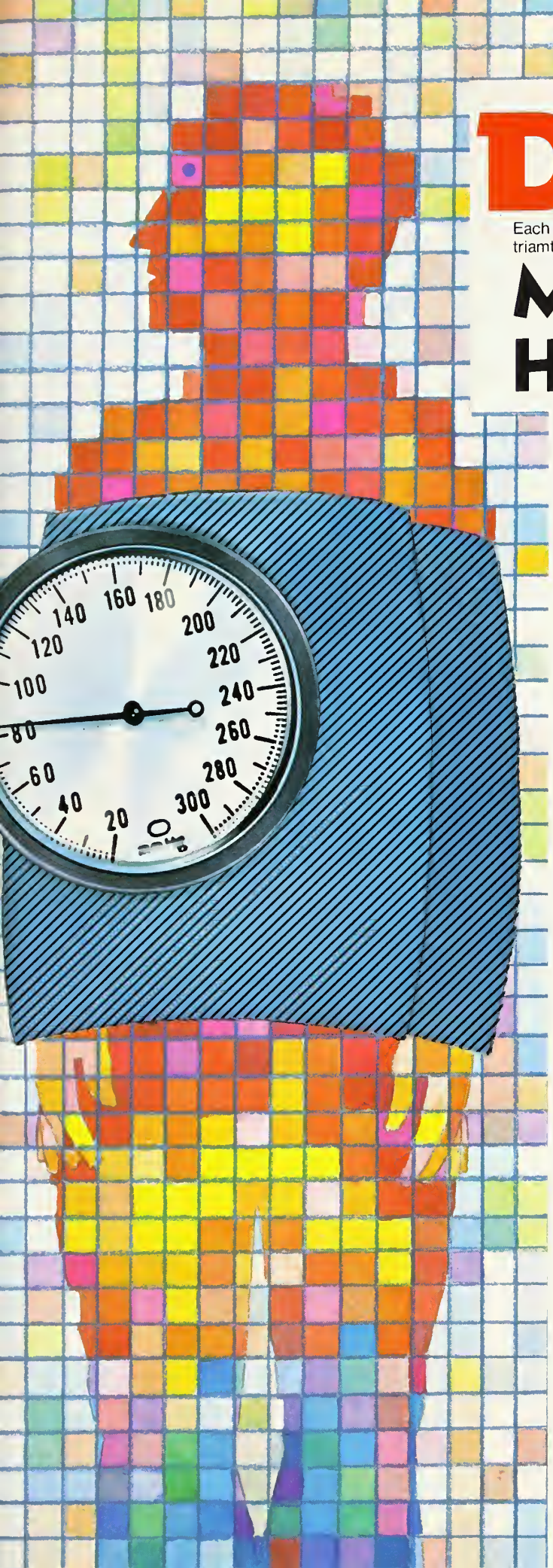
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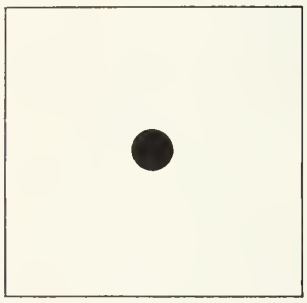
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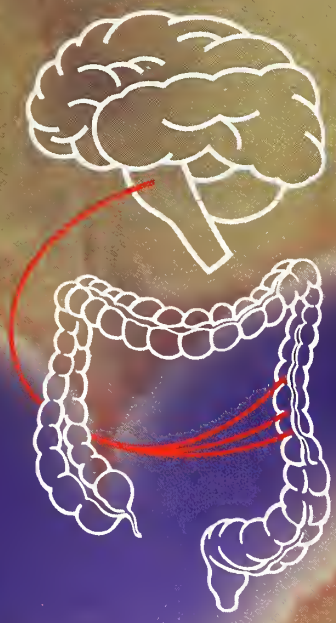
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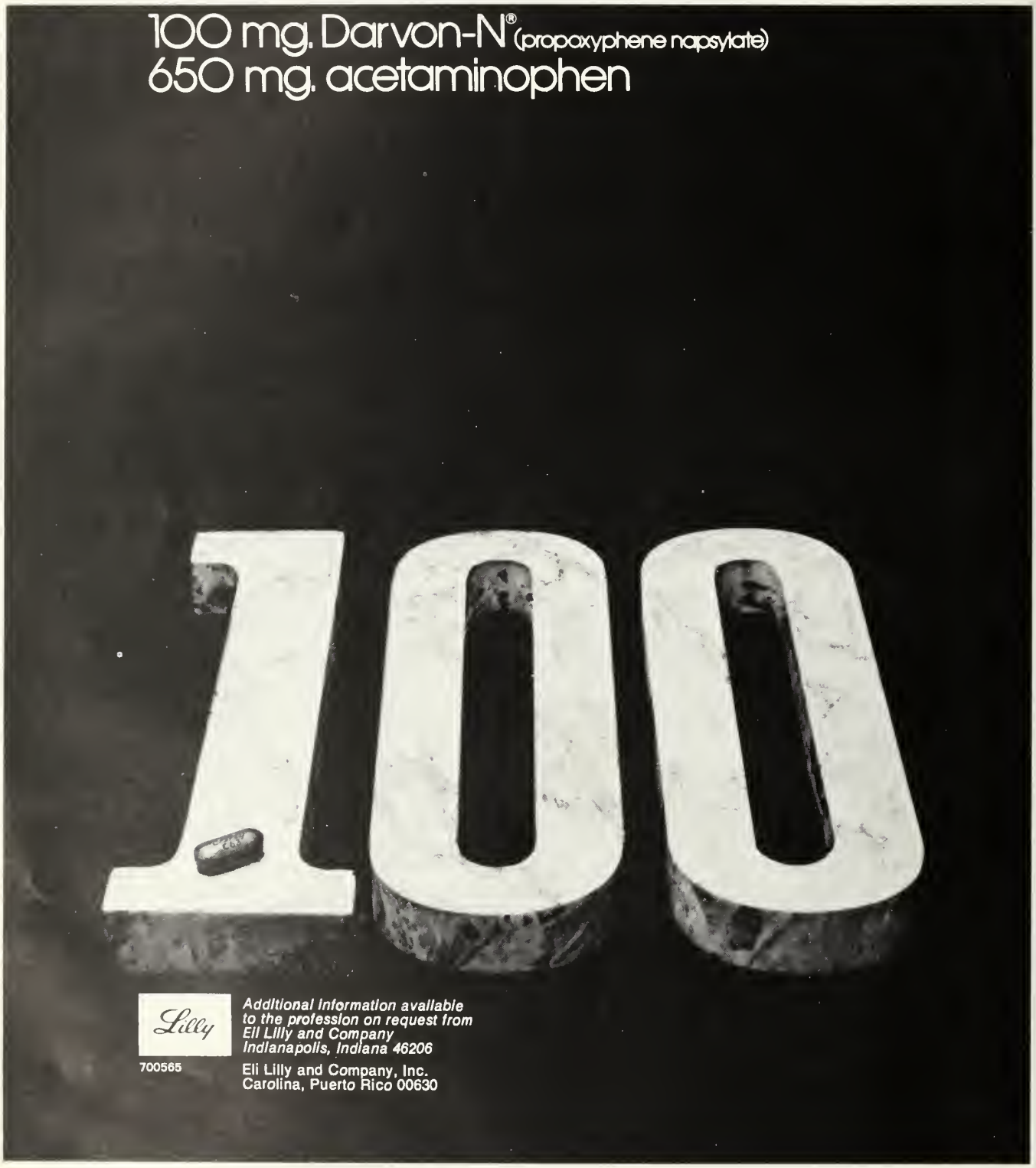
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EDITORIALS

Sigurd Walter Johnsen, M.D.—1895-1978

On December 26, 1978, Doctor Sigurd W. Johnsen, 159th President of the Medical Society of New Jersey, died, at the age of 83.

Born in Chicago, Dr. Johnsen received his early education there and was awarded the degree of Bachelor of Science by the University of Chicago in 1921. He pursued his medical studies at Cornell University, which conferred upon him his doctorate in medicine in 1925. He interned first at Jersey City Hospital, and in 1926 completed a rotating internship at Passaic General Hospital. In that year he opened his office for private practice in Passaic, which he maintained until retirement in 1972. He did postgraduate work in gastroenterology and radiology at Columbia University. Subsequently he became a Fellow of the American College of Physicians, of the American College of Gastroenterology, and of the Radiological Society of North America.

Dr. Johnsen was a founder—and for more than a decade President—of the Passaic-Bergen Medical-Dental Service Bureau. In 1941, he was elected President of the Passaic County Medical Society.

His earliest official service as a member of the Medical Society of New Jersey began in 1937, when he was appointed to the Welfare Committee. In 1939 he served as Chairman of the Committee on Auxiliary Medical Services. In 1943 he became Chairman of the Committee on Medical Practice, and two years later assumed Chairmanship of the Welfare Committee. He was elected to the Board of Trustees in 1946, and in 1948 became Second Vice-President. He served as the Society's President in 1951-1952. A man of singular earnestness and of conscientious and productive industry, Dr. Johnsen exercised a salutary and stimulating leadership in all the existing offices which his colleagues entrusted to him.

He is survived by his wife, the former Aletta Johnson; a son, Sigurd E. Johnsen, M.D., of Montclair; a daughter, Mrs. Robert (Miriam) Chamberlain, of West Hartford, Connecticut; a brother, Thilo, and a sister, Mrs. Gladys Wilson, both of Chicago; and five grandchildren.

Richard I. Nevin

Obesity and Diabetes

In the most definitive text on the epidemiology of diabetes, West* points out that careful studies have shown the rates of diabetes in lean populations are "one-third to one-tenth as great as in genetically related societies where corpulence is common." He also emphasizes that the "familial aggregation of diabetes is secondary to the familial aggregation of obesity." We all have known that obesity is a precipitating factor of diabetes in those persons who are genetically susceptible, but West* believes new data "strongly suggest that obesity alone is quite capable of producing diabetes, and often does."

In this issue (page 175), Kirschner and his associates discuss a successful method for the control of major obesity. Among their study group were 48 patients with previously diagnosed diabetes. Of this group, 20 were using insulin and 28 were taking oral hypoglycemic drugs. All patients receiving oral hypoglycemic agents regained normal carbohydrate tolerance after weight reduction and 18 of the 20 patients receiving insulin were able to discontinue their daily injections entirely within one week of starting the supplemented fast program. Although the weight loss alone in one week would not explain this phenomenon, a salutary effect on insulin receptors, so as to alleviate the insulin resistance of obese diabetics, would. As indicated by the authors, improvement in hypertriglyceridemia was seen in the diabetic patients along with the improvement in carbohydrate tolerance.

Although the application of the method described by the authors to the control of obesity in clinical office practice on a univeral basis may not be imminent, there is little doubt that weight reduction by any method would be beneficial for the person with diabetes.

Insulin-secretion studies have shown that beta-cell function in diabetics often returns to normal after sustained alleviation of obesity. West states "that a preventive and a cure (of diabetes) are already at hand for most diabetes." "The cause is usually obesity; the preventive, and often the cure, is leanness."*

Diabetes mellitus is a model chronic disease which seems to lead to truths worthy of wide application. If preventive medicine is to become a universal reality, diabetes can be the springboard for such a demonstration. Obesity leads to diabetes. Leanness alleviates diabetes in a majority of cases. Prevention of obesity can prevent diabetes. A.K.

*West KM: *Epidemiology of Diabetes and Its Vascular Lesions*. New York, Elsevier North-Holland, Inc., 1978, pp. X, XI.

The Clasped Hands

Our cover photograph clearly depicts the professional life and the warmth and humanity of a former chairman of the Board of Trustees of MSNJ, David Eckstein (see personal note, page 225, this issue). More than that, the picture sums up what computers, complex x-ray and laboratory equipment, allied health professionals, and do-it-yourself medical books cannot replace—the relationship between one patient and one doctor. There is trust and gratitude in the face of that beautiful white-haired lady.

The clasped hands—one pair gnarled and arthritic and the other skilled in the tactile and percussive arts of medicine—suggest the basic reasons that most of us entered and continue in the practice of medicine. "To do good for humanity" is the most frequent reason medical school applicants give for their desire to become physicians.

Despite the rampant charges foisted on physicians and the unholy intrusions by government bureaucrats, politically inspired consumer advocates, and others, the physician who can grasp the hands of his patient and transfer his concern for that patient's well-being has nothing to fear in the future.

A.K.

PSRO Update

The New York Times on Wednesday, November 9, 1977, published an article entitled "Efficacy of Review by Medical Teams Challenged in Study." In this article it was related that the Health Services Administration reported Professional Standards Review Organizations (PSROs) as not now cost effective. Well-intentioned members of PSRO organizations became discouraged and morale appeared to slip with both administrative and professional members of PSRO groups, in spite of excellent organization and many long hours by dedicated professionals. It appeared that the Government may find cost effectiveness to be a common denominator for longevity of a program which many of us felt to be the last ditch defense for private practice of medicine.

Recent events appeared to give hope for continuation of activities, and among the many reasons for this resurgence is a renewed acceptance by the physicians themselves of the importance of PSRO activities. Instead of hearing, "We have seen the enemy and he is us," either in jest or on a serious level, we are now beginning to hear comments that "You people are really on our side; you are practicing, active physicians who are professionally up to 'snuff'; you are really trying to help us maintain high levels of medical quality."

Within three months, national returns indicate that length of stay is decreasing and quality of care is on the upswing.

In the northeast the average length of stay has been cut by 2.6 days, and both physicians in the field and PSRO physician advisors are talking quality of care. Medical care evaluation (MCE) studies have indicated that guidelines are being drawn, suggesting directions of care that doctors themselves wish to be part of, and no less a prominent organization than the American Medical Association, in the *American Medical News* published December 15, 1978, expressed strong support "for continuation of the PSRO program as a professionally directed effort to ensure that care provided to patients is of high quality, appropriate duration, and is rendered in an appropriate setting at a reasonable cost."

The PSRO program is under constant surveillance and we physician members continuously are monitoring new methods of patient care review and assurance of quality controls.

We physicians should remain as one voice. It is important for the physician to realize that he and his PSRO are still in control of private practice and now have received the blessings of both government agencies and the American Medical Association.

Nelson Manowitz, M.D., Essex Physicians' Review Organization
Donald P. Burt, M.D., Area I-Region II PSRO
Charles E. Dooley, M.D., PSRO of Union County
William A. Dwyer, M.D., Passaic Valley PSRO
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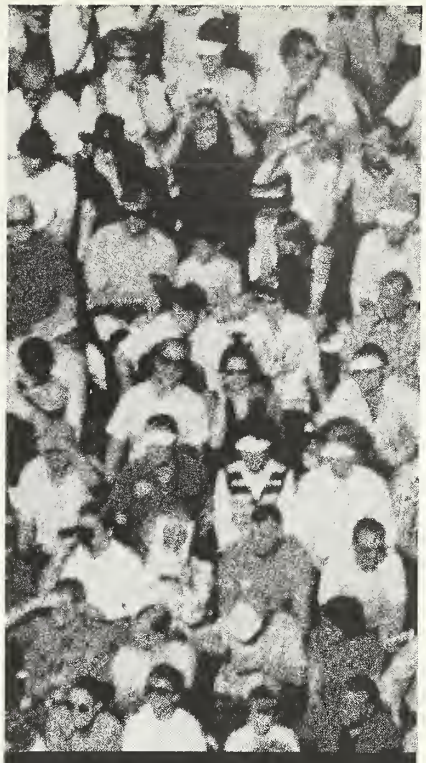


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*The safety and effectiveness of Motrin have not been established in patients with Functional Class IV rheumatoid arthritis (incapacitated, largely or wholly bedridden, or confined to wheelchair; little or no self-care).

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Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

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Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin (ibuprofen) is gastrointestinal (4% to 16%). This includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness*, headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

Incidence: Unmarked 1% to 3%; *3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

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Supplemented Starvation: A Successful Method for Control of Major Obesity*

MARVIN A. KIRSCHNER, M.D., Newark
GEORGE SCHNEIDER, M.D., East Orange
NORMAN ERTEL, M.D., East Orange
GONZALO CORTES, M.D., Newark

The results of the first year's experience with a semi-starvation method of weight reduction for morbidly obese patients is presented. Using this approach, 77 percent of all men and 45 percent of all women entering the program lost 40 pounds or more. Associated medical problems such as hypertension, diabetes mellitus, and hypertriglyceridemia were benefited significantly by this method alone.

Obesity is well established as a major health problem in our society.[1] The association of obesity with coronary artery disease, hypertension, hyperlipidemia, diabetes mellitus, and endocrine-related neoplasms is well known. Mortality rates rise precipitously with increasing body size.[2]

In view of the urgent health hazard presented by widespread obesity, we have adapted a supplemented starvation program for control of the seriously obese adult patient.[3,4] The results of our first year's experience with out-patient management of a supplemented starvation program is presented in the current report. They suggest that a method finally is available for control of a problem which heretofore has been refractory to the usual dietary measures.

METHODS

A modification of the supplemented starvation regimen of Genuth and Vertes was used for the purpose of rapid weight reduction.[3,4] All patients were evaluated thoroughly prior to entering into the program, with extensive medical history and physical examination, electrocardiogram, and serum chemistry measurements consisting of CBC, SMA-12, electrolytes, and triglycerides. Thyroid function tests were performed where indicated. Following this initial evaluation, patients were instructed to stop all food, except for coffee, tea with lemon, or diet sodas. A regimen consisting of five feedings per day of Optifast® (Delmark Corporation, Minneapolis, Minn.) was provided.[4] Optifast® is a powder, providing a daily intake of 300 calories from the following constituents:

(1) 45 gms protein (from pasteurized egg white solids)
(2) 30 gms sucrose
(3) 0.6 gms calcium
(4) 0.35 gms phosphates
(5) 18 mgm iron
(6) 150 mgs magnesium
(7) 2.0 mgm copper
(8) 15 mgm zinc
(9) 860 mgm potassium
(10) 920 mgm sodium
(11) 40 mgm manganese

The powder is blended with any cold non-caloric beverage such as water, diet soda, and so on. To further supplement the above preparation, patients were given 20Meq/day potassium and were instructed to take one multivitamin tablet daily. All diuretic drugs were discontinued at the start of the supplemented starvation program.

Instructions for daily isometric exercises were provided and patients were encouraged to engage in athletic activities and exercises to which they already had been accustomed. New or heroic athletic programs were discouraged. Patients

*From the Metabolic and Nutrition Service, Newark Beth Israel Medical Center, the Veterans Administration Hospital in East Orange, and New Jersey Medical School, Newark. Dr. Kirschner may be addressed at Newark Beth Israel Medical Center, 201 Lyons Avenue, Newark, 07112.

were maintained on this regimen until ideal body weight was achieved. During this time they were followed closely on a weekly basis and seen each visit by their treating physician. Dietary retraining was begun and psychological counseling was offered. Blood samples were obtained every other week and monitored for CBC, SMA-12, electrolytes, and triglycerides. Repeat electrocardiograms were performed as indicated.

Patients with significant preexisting illnesses or following complex drug schedules were admitted to the hospital for a one-week period in order to start the program. During this time, hypertensive patients were weaned off diuretics; other anti-hypertensive drugs were reduced. Diabetic patients were monitored carefully during the induction of the starvation; insulin was discontinued entirely during the first week. Patients with stable preexisting heart disease also were monitored carefully. A cardiologic consultation was obtained to be certain that the starvation would not aggravate their underlying problem. Patients with Pickwickian syndrome similarly were monitored during the induction of the starvation. Following this five-to-nine-day period of hospitalization, patients were discharged and followed on an outpatient basis, as above.

PATIENTS

During the first year of this program, 480 patients were registered. Women outnumbered men by a ratio of five to one. The occupational background of the patients engaged in this program was as follows:

Professional: lawyers, physicians, teachers	45%
Housewives	19%
Office and hospital personnel	15%
Blue collar jobs	10%
Other	11%

The clinical features of the patients in our program are listed in Table I. The average weight of male patients was 265 pounds and that of women was 225 pounds. Both groups of patients averaged 95 pounds above ideal body weight as judged from N.Y. Metropolitan Life Insurance tables for respective sex and body size, indicative of the fact that this population was indeed morbidly obese. The prevalence of associated medical abnormalities was striking, considering that the mean age for men was 36 and for women 37.

Table I
Clinicol Features of 480 Patients in a Supplemented Fasting Program

	Average weight (Pounds)	Maximal weight (Pounds)
1) Initial Weight		
Men (80)	265	535
Women (400)	225	390
2) Associated Medical Problems		
Hypertension	40%	
Diabetes Mellitus	10%	
Hyperlipidemia	67%	
Triglycerides	45%	
Cholesterol	22%	

RESULTS

(1) **Weight loss**—On this regimen of supplemented starvation, patients lost weight at a gratifying rate (Figure 1). During the initial week, men lost an average of 11.0 pounds (range 5-29) and women lost an average of 7.7 pounds (range 4-22). Following this initial natriuretic period, men lost weight at an average of 4.2 pounds per week and women lost at an average of 3.0 pounds per week. In general, patients with greater initial weights lost weight more rapidly than those with lower starting weights. No "plateau periods" were observed. In most cases, failure of a patient to lose the expected weight was caused by non-compliance.

At the time of this tabulation, the average patient has been in the program for 14 weeks. Mean weight losses for the program averaged 54.9 pounds for men and 44.3 pounds for women. To date 43 patients have lost 100 pounds or more and one patient has lost 230 pounds over a nine-month course (Figure 2).

Not all patients were able to tolerate the rigors and discipline of a supplemented starvation program. Indeed 70 patients (16 percent) dropped out of the program within the first three weeks and are not considered in the long-range statistics of success/failure.

(2) **Effects on Underlying Medical Problems**—The results of significant weight loss in terms of amelioration of associated diseases are presented in Table II.

(a) **Hypertension:** On entering the supplemented starvation program, 192 patients were hypertensive (greater than 140/90 mmHg). Of these, 123 were on some form of anti-hypertensive drug at the outset (60 percent), indicating more importantly that 40 percent of our population was either unaware of this medical problem or failed to adhere to various prescribed therapies.

As the result of weight loss, 77 percent of patients on anti-hypertensive drugs were able to discontinue these medications because of normalization of blood pressure. An additional 23 patients achieved normal blood pressures but still required anti-hypertensive medications, albeit at a lower dose. Only eight patients of the starting group had persistent

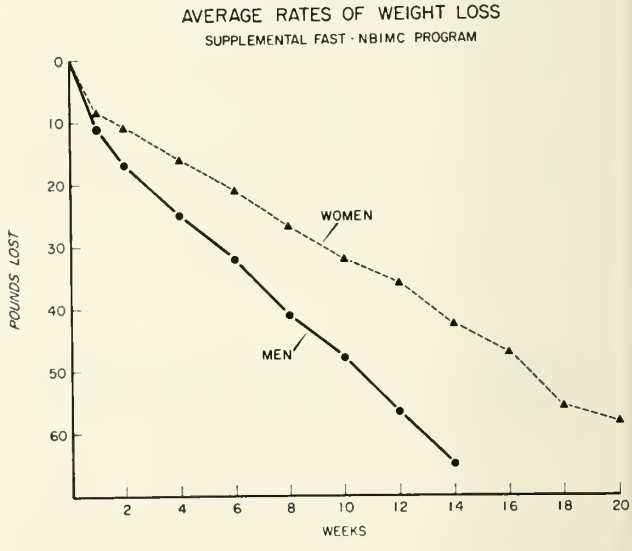


Figure 1—Average rates of weight loss achieved during supplemental starvation. During the initial week, men lost an average of 11.0 pounds and women an average of 7.7 pounds. In subsequent weeks men lost an average of 4.2 pounds and women an average of 3.0 pounds per week.

Table II
Effects of Weight Loss on Underlying Medical Problems

1) Hypertension	
BP normalized, all drugs stopped	72%
BP normal with drugs	21%
BP still high on drugs	7%
2) Diabetes Mellitus	
Discontinued oral hypoglycemic drugs	100%
Discontinued insulin	90%
3) Hypertriglyceridemia	
Normalized by diet	65%
Lowered by diet, but still abnormal	35%

hypertension despite weight loss and medication. Thus, of the hypertensive population, 93 percent had return of their blood pressure to normal either by dieting alone, or in combination with anti-hypertensive drugs.

(b) **Diabetes Mellitus:** Forty-eight patients with previously diagnosed diabetes mellitus entered into the program. Of these, 28 were receiving oral hypoglycemics and 20 were on varying doses of insulin ranging from 20 to 150 units per day.

As noted in Table II, all patients receiving oral hypoglycemic drugs experienced amelioration of their blood glucose abnormalities by dieting alone and were able to discontinue their drugs. More impressive was the observation that of the 20 patients initially receiving insulin, 18 were able to discontinue this medication entirely within one week after starting the supplemented fast. The remaining two patients required a lesser dose but could not be weaned entirely from exogenous insulin.

(c) **Hypertriglyceridemia:** Two hundred eighteen of the 480 patients entering into the program had elevated fasting serum triglyceride values.Of this group, 142 (65 percent) had normalization of triglycerides by dieting, 76 (35 percent) had lower, but still abnormal values. All patients experienced significant decreases in their serum triglycerides. Changes in serum cholesterol were of considerably less magnitude.

(3) **Complications**—A tabulation of side effects and complications noted during the first year with the supplemented fasting program is presented in Table III. The most common problems noted were postural lightheadedness and mild (late) scalp hair loss. The latter was seen in 37 patients, but in no case was the alopecia of sufficient magnitude to interrupt the diet. In all cases, this problem was reversed within several months after the patients were re-introduced to normal diets. Acute gouty attacks were noted in only three patients, although hyperuricemia occurred frequently during the first month of the fast and was controlled by allopurinol. Transient peroneal nerve palsy was noted in two patients.

During the first year of the program, two deaths were recorded. Both were felt to be unrelated to the fast: One patient succumbed to a ruptured dissecting abdominal aneurysm. The other was a 52-year-old male (with a history of a previous myocardial infarction) who lost 80 pounds over four months on the program. He then suffered a myocardial infarction. He was taken off the program and an uneventful recovery ensued. He remained off the program for four months, and then suffered a third, fatal myocardial infarction.

DISCUSSION

Obesity represents a major health hazard in the United States as well as in other nations.[1] Keys estimated that 63

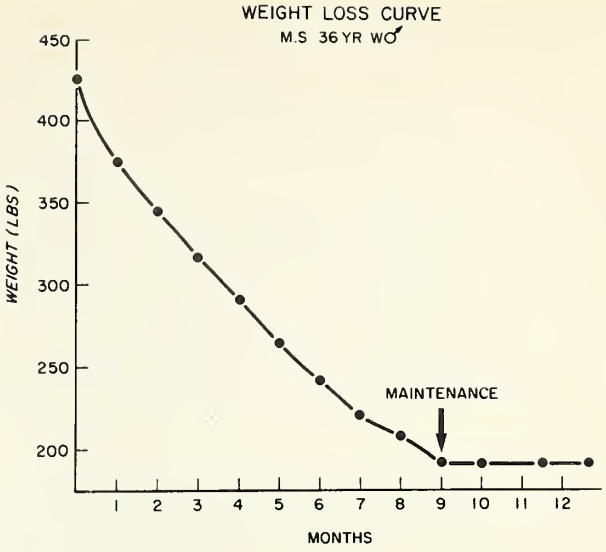


Figure 2—Weight loss curve in a 36-year-old man. After 9 months of supplemental starvation, a total loss of 230 pounds was achieved. Successful maintenance has now been extended for eight months.

percent of adult Americans were obese, judging from skin fold measurements.[5], [6] Further, the problem appears to be worsening. In 1956, 16.4 percent of American men were classified as obese (15 to 25 percent above ideal body weight) or severely obese (greater than 25 percent above ideal body weight). In 1971 this figure increased to 29.0 percent. In women, the figures are even more ominous. In 1956, 42 percent were classified as obese or severely obese; in 1971, 46.8 percent of American women were obese with a greater increase in the severely obese group.[7] The medical complications of obesity include accelerated arteriosclerotic cardiovascular disease, hypertension, diabetes mellitus, hyperlipidemia, gallbladder disease, and endocrine-related neoplasms and are well known. In general, obesity appears to accelerate the degenerative diseases of aging.

In view of the increasing incidence and prevalence of obesity in our society, various schemes have been devised over the years for the treatment of obesity. Almost all weight loss schemes involve caloric restrictions with varying adjunctive measures including anorectic drugs, metabolism-accelerating drugs, peer pressure, psychological counseling, or hypnosis. In some cases, shifts of dietary components were used for short-term benefits. One scheme for promoting major weight loss includes intestinal or gastric bypass surgery to decrease the gastrointestinal absorptive surface. Of the various regimens, caloric restriction, either with no counseling, individual counseling, or with peer pressure or self-help groups, has been most widely used. Unfortunately, the success rate of calorie restriction regimens has been extremely low. As noted by Bray,[8] the success rate of calorie-restriction programs for loss of 40 pounds or more is only four to ten percent.

In recent years, there has been a renewed interest in the use of starvation as a method of weight loss. Earlier observations showed that starvation promotes ketosis within a few days. Elevated blood ketones not only provide the major energy source but also produce anorexia which aids in continual adherence to the diet.[9] Genuth and Vertes[3,4] as well as Bistrian and Blackburn[10] reported that supplemented starvation with 40 to 100 grams per day of essential proteins and amino acids reduces the negative nitrogen balance of total

Table III
Clinical Side Effects of Supplemental Starvation

Postural Lightheadedness
Transient alopecia
Cold intolerance
Halitosis
Dry skin
Menstrual irregularities
Acute gout
Foot drop
Acute psychosis
Fatigue

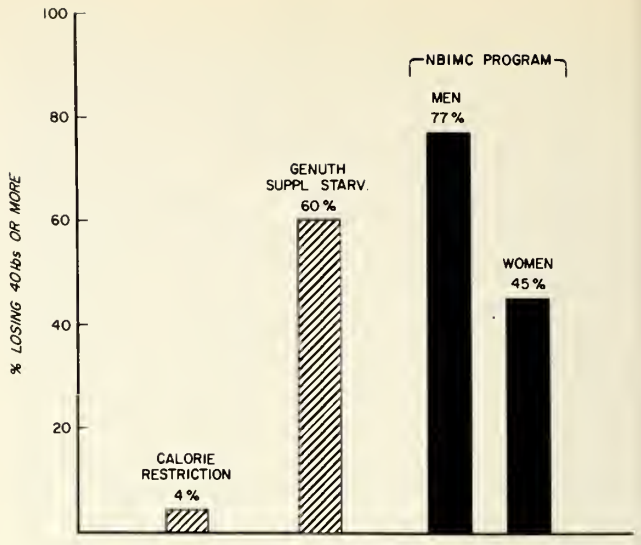


Figure 3—Successful rates of various programs for achieving major weight loss. Calorie-restriction program (after Bray[8]) is quite poor. The success rate of our program is contrasted with that of Genuth *et al.*[3,4] See text for details.

starvation. Whereas Bistrian derived this regimen using weighed amounts of table foods,[10] Vertes and Genuth[4] adopted an egg albumen formulation which removes any decision-making by the patient. Vertes further added minimal amounts of sucrose to the artificial regimen in order to provide baseline carbohydrate to decrease some of the observed blunting of higher order mentation noted during severe ketosis. In carefully controlled supplemental starvation programs, patients thus continue to receive exogenous essential proteins, carbohydrates, and a total of 300 calories per day.

Metabolic effects of supplemental starvation include:

(1) Brisk natriuresis which accounts for the large weight loss during the initial week. Occasionally this produces volume depletion and transient lightheadedness over the first weeks.

(2) Hyperuricemia occurs frequently and needs careful monitoring. Occasionally allopurinol is required to prevent acute gouty attacks.

(3) Hypokalemia may develop since intake of potassium is not sufficient to replace urinary losses. Since serum potassium is a poor index of total body potassium, patients are prescribed extra potassium at the outset rather than waiting for chemical or clinical hypokalemia to develop.

Using the supplemented starvation approach, Vertes and Genuth reported an extraordinary success rate for the treatment of severely obese patients heretofore refractory to other measures.[3], [4] Data from the Cleveland Group appear to be holding firm after five years.

The Newark Beth Israel Program is fashioned after that of Vertes, Genuth, and Hazelton.[4] Seventy percent of the male patients and 45 percent of the women successfully lost 40 pounds or more. Our figures compare favorably with those of Genuth and are contrasted with the poor success rates of calorie-restriction programs, as shown in Figure 3. Indeed a substantial number of patients have lost 100 pounds or more. The usefulness of such a program is further emphasized by the fact that all patients are able to lead normal lives and carry on full work and athletic loads during the weight loss process with a feeling of well-being.

Amelioration of underlying medical problems was by far the most gratifying aspect of the supplemental fasting program.

(1) **Hypertension** was readily controlled by diet and weight loss. Seventy-seven percent of hypertensive patients were able to discontinue all blood pressure medications. Only eight percent of our hypertensive group were refractory to diet and drugs, confirming the studies of Reisen that diet alone is an effective method for control of hypertension.[11]

(2) **Diabetes Mellitus** in obese patients was most readily controlled by starvation. Oral hypoglycemic agents were discontinued in all 28 patients without difficulty. However, we were most impressed that in 18 of 20 patients insulin requirements as high as 150 units per day could be discontinued completely within one to four days of starting the fast, confirming the work of Archer that starvation liberates insulin receptor sites making endogenous insulin fully effective in controlling blood sugar.[12]

(3) **Hypertriglyceridemia** was reduced significantly by dieting in all patients. Of the 218 patients with elevated triglycerides, two-thirds experienced normalization over a period of weeks as the result of the starvation regimen. Although cholesterol levels were lowered in most patients, the changes were far more modest.

As for complications, these were minimal and frequently intercepted at an early stage. Transient alopecia and postural lightheadedness were the most common complaints. Foot drop (peroneal neuropathy) has been reported by Vertes and Genuth;[13] in our cases the problem resolved over several weeks. Other lesser complaints included halitosis, menstrual irregularities, and cold intolerance.

In view of the marked nationwide concern over cardiovascular deaths and liquid protein diets,[14-16] we were most concerned with this potential complication in our supplemented-starvation program. To date, two patients registered in the program have died, both of pre-existing problems felt to be unrelated to the diet. In the Cleveland program, extending over a five-year period, there also appears to be no increased incidence of unexpected cardiovascular deaths.[17] At the present time it has been estimated that 80,000 to 100,000 patients are registered at various "Optifast®" programs through the country with no acceleration of cardiovascular complications.

It thus appears that a supplemented fasting outpatient program, carefully controlled by physicians with experience and training in metabolic disorders, is an effective and safe method for controlling a medical health problem which heretofore has been refractory to other measures.

SUMMARY

Morbid obesity represents a major health hazard today. Until now, conventional approaches to the treatment of this disorder have been uniformly unsuccessful. We currently are presenting the first year's experience with a semi-starvation approach to weight reduction. Seventy-seven percent of all men and 45 percent of all women entering the program lost 40 pounds or more. Nine percent of all patients lost at least 100 pounds. After the initial week's diuresis, men lost an average of 4.2 pounds per week and women lost an average of 3.0 pounds per week. Seventy-seven percent of all hypertensive patients were taken off their anti-hypertensive medication and had a normalization of blood sugar without medication. Sixty-five percent of patients with hypertriglyceridemia had a normalization of their triglycerides with weight loss. Side effects or complications of this regimen were minimal.

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[1] Tinkelman, D.G., Carroll, M.S., Vanderpool, G., Jones, M. The bioavailability of theophylline in elixir and micro-pulverized forms. Medical Challenge 10:24-26, 1978.

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Hypotensive Effect of Increased Dietary Linoleic Acid in Mildly Hypertensive Humans

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Increasing dietary linoleic acid was associated with a decrease in both systolic and diastolic blood pressure within one month in 28 mildly hypertensive adults. Concomitant with the blood pressure decrease, a decrease in serum cholesterol and improved platelet function were observed.

Iacono *et al.*[1] observed that a linoleic acid-enriched, cholesterol-lowering diet appeared to exert a hypotensive effect in man. In a preliminary study on eight mildly hypertensive adults, Comberg *et al.*[2] were able to demonstrate a 10 mmHg decrease in diastolic blood pressure in three weeks. The diastolic blood pressure returned to the pre-experimental elevated level three months after terminating the diet. Lee[3] has shown that prostaglandins, of which linoleic acid is the ultimate biological precursor fatty acid[4,5] induce increased sodium and water excretion and peripheral arteriolar dilatation.

A study was undertaken to expand upon these preliminary observations and to determine whether the linoleic acid composition of the diet of a free-living population can be increased sufficiently to observe both the hypotensive and hypolipemic effects attributed to increased dietary linoleic acid. Additionally, since increased linoleic acid is known to decrease platelet aggregability[6-8] and since platelets have been reported to exhibit increased aggregability in hypertension, [9,10] the effect of the increased linoleic acid upon platelet function was evaluated.

MATERIALS AND METHODS

The patient population consisted of 28 consecutively selected adults, 19 women and nine men, with a mean age 63.2±2.0 years. They were either residents in a senior citizens' housing project or were patients of cooperating physicians in Essex County, New Jersey, and who met the study criteria. They were all mild hypertensives (BP 180/105 or less), free living, and with no acute disease process that would confound the platelet or blood pressure data; they had no chronic illness requiring the use of salicylates. Of the 28 study volunteers, three were controlled adult onset diabetics, two had suffered a myocardial infarction sometime in the past, and one was a diabetic who had suffered a myocardial infarction. Lipoprotein phenotyping at the time of entry into the study indicated that of the 28 volunteers, two were Fredrickson lipoprotein phenotype II_A, two were phenotype II_B and five were phenotype IV. No phenotypes I, III or V were found. In order not to confound the study, all volunteers were either on no medication or, if they were on diuretic medication they were taken off this medication by their physician one month prior to the study. They remained off the medication for the period of the study.

Prior to starting the study, a blood pressure measurement and a platelet function determination were made. A 14-hour fasting serum specimen and a morning urine sample were obtained for analysis. The volunteers were placed on a linoleic acid-enriched diet for four weeks. They were supplied with a sunflower oil-based margarine (54% linoleic acid),

*From the Division of Community Health Services, New Jersey State Department of Health. Supported in part by grants from the New Jersey State Department of Health and the Charles Edison Fund. Dr. Fleischman may be addressed at that office, 48 Plymouth Street, Montclair, New Jersey 07042.

Table 1

Effect of Increased Linaleic Acid in the Diet upan Blaad Pressure, Platelet Functian, Blaad and Urine Canstituents in 28 Hypertensive Adults

Measurement	Unitage	Preexperimental	One Manth an Diet	One Month aff Diet
Systalic Blaad Pressure	Torr	147.6 ± 2.55	139.5 ± 2.05**	136.5 ± 2.24
Diastalic Blaad Pressure	Tarr	91.0 ± 1.58	86.2 ± 1.50**	85.2 ± 1.72
		Platelet Functian		
Aggregation Time	Secands	428 ± 37.9	539 ± 24.6*	430 ± 40.5
Disaggregatian Time	Secands	105 ± 29.0	70 ± 8.5	107 ± 29.9
		Blaad Chemistry		
Dietary Linaleate	% Energy	4.0 ± 0.30	5.2 ± 0.42**	4.1 ± 0.44**
Serum Chalesteral	Mg/dl	224.0 ± 6.86	210.1 ± 6.20**	217.8 ± 8.94
Serum Triglycerides	Mg/dl	108.8 ± 11.38	116.2 ± 13.84	105.3 ± 9.10
Creatinine	Mg/dl	0.88 ± 0.03	0.98 ± 0.04*	0.96 ± 0.03
Sodium	mMale/l	142.8 ± 0.62	140.8 ± 0.56**	140.3 ± 0.75
Patassium	mMale/l	4.71 ± 0.11	4.56 ± 0.09	4.48 ± 0.09
Osmalality	mOsmales/l	298.9 ± 3.23	306.5 ± 5.26	294.7 ± 1.42*
		Urine		
Sadium Excretian	mMale/mg Creatinine	0.11 ± 0.01	0.09 ± 0.01	0.10 ± 0.01
Patassium Excretian	mMale/mg Creatinine	0.05 ± 0.006	0.03 ± 0.004**	0.04 ± 0.003
Osmotic Clearance		2.22 ± 0.18	2.00 ± 0.16	2.43 ± 0.17**

[1]Mean ± Standard Errar af the Mean

**P 0.01 by paired t test compared ta immediately preceding measurement

* P 0.05 by paired t test campared ta immediately preceding measurement

Osmatic Clearance defined as: Osmalality af Urine/Osmalality af Serum Reference Range abave 1.50

sunflower oil (65% linoleic acid), and filled cheese. Other than increasing the linoleic acid content of the diet, no attempt was made to alter other normal eating habits, particularly in relation to sodium chloride intake. After one month on diet, all measurements were repeated, and the volunteers reverted to their normal diets for an additional month. At the end of the month off diet, all measurements were repeated one final time. After the last measurement, the patients were lost to additional follow-up diet since their respective physicians put them back on either diet or medication.

Platelet function was measured by the filtragometer procedure [6-8]. This procedure measures the aggregation of platelets in flowing blood and is assessed by drawing blood via an 18 gauge 1.5 inch disposable needle from an antecubital vein through a microfilter of 20 mm pore size into a motor-driven syringe at a constant rate of 2.0 ml per minute. This filtration method is based on the use of a pore size which permitted the passage of red and white cells and platelets but was occluded by platelet aggregates. The entire system was siliconized and maintained at 37°C. Heparin was infused into the system proximal to the filter at an anticoagulant concentration of five units per ml blood. Pressure was monitored proximal and distal to the filter, the rise in pressure difference reflecting occlusion of the filter by aggregated particles. The aggregation time was defined as the time in seconds required to reach a gradient of five mmHg. Following aggregation, the heparin infusion was discontinued and a 38 percent sodium citrate solution was infused at a rate of 0.1 ml per minute. Disaggregation time was defined as the time in seconds required to clear the filters as shown by a reversal of the pressure gradient. To check the patency of the needle, the pressure between the needle and the filter was monitored continuously. Blood pressures were measured in the sitting position three times in succession at five minute intervals. No smoking was permitted for at least 90 minutes prior to the measurement since this was known to affect both blood pressure and platelet function measurements.[11] Serum and urine chemistries were done by standard automated methodologies. As an indicator of dietary adherence, the fatty acid composition of the serum triglycerides was measured by gas liquid chromatographic analysis.[12] Statistical analysis was done by the two-tailed, paired student "t" test.

RESULTS

Systolic blood pressure decreased from a preexperimental level of 147.6±2.6 to 139.5±2.1 torr after one month on diet, $p<0.01$ (Table 1). Similarly, diastolic blood pressure decreased from 91.0±1.6 to 86.2±1.5 torr, $p<0.01$, concomitant with an increase in dietary linoleate of 1.2 percent of energy (percent of calories), $p<0.01$. One month after going off the diet, blood pressure remained unchanged while the linoleic acid concentration of the diet returned to its preexperimental level.

Concomitant with the increased dietary linoleic acid ingestion, platelet aggregation time increased from 428±37.9 to 539±24.6 seconds, $p<0.05$, while disaggregation time decreased from 105±29.0 to 70±8.5 seconds. One month after diet, both values returned to their preexperimental levels. Serum cholesterol decreased 14 mg/dl while on diet, $p<0.01$, and increased toward the preexperimental level after cessation of the diet. There was a decrease in serum sodium on diet, $p<0.01$, but not in serum potassium or triglycerides.

No significant alteration was noted in sodium intake during the study period as evinced by excretion determination but potassium excretion was decreased while on diet, from 0.05±0.006 to 0.03±0.004 M Mole/mg creatinine while on diet, $p<0.01$. There was also no significant weight change in the volunteers while on the study. On the normal diet, their mean weight was 77.7±2.8 kg and 77.7±2.7 kg on the linoleic acid enriched diet.

DISCUSSION

The decrease in blood pressure noted on the linoleic acid-enriched diets is in agreement with that reported by Iacono *et al.*[1] and Comberg *et al.*[2] in man and Triebe *et al.*[13] and Ten Hoor and Van de Graaf[14] in rats. Although Triebe *et al.*, Ten Hoor and Van de Graaf, and Iacono *et al.* employed higher dietary linoleic acid concentrations of 14 to 22 and 6 to 8

percent of energy respectively in their studies, the level of linoleic acid in the diet noted in this study was similar to that found in the previous studies in this area.[6,7].

As might be expected from previous studies[6-8] as dietary linoleic acid increased, platelets became less aggregable and serum cholesterol decreased. When the volunteers reverted to their normal diet which contained a lower linoleic acid concentration, serum cholesterol increased and the platelets became more aggregable.

The failure of the blood pressure to increase within one month after going off the linoleic acid-enriched diet cannot be explained at this time. The possibility of a conditioning effect being present certainly has to be considered. However, since all of the volunteers had blood pressure elevations known and under some management previously for some time, this explanation does not appear to be likely. Comberg *et al.*[2] measured blood pressure three months after diet and found the blood pressure increased. Even three months after diet, while the diastolic blood pressure had increased, it was still lower than the value they reported both six months prior to the diet and the preexperimental value. The possibility exists that a lag effect of polyunsaturated fatty acid release from depot fat which has been noted in cross-over feeding studies on serum cholesterol levels in this laboratory may be operative in regard to blood pressure levels also. It had been noted to last for as long as a period of three weeks and could be the explanation for the failure of the blood pressure to return to preexperimental level one month after diet cessation. In the present study, since all volunteers were under the care of their own private physicians, they were lost to longer terms follow-up since they were put back on medication as soon as the one month off diet measurement was completed.

This study, while larger than the preliminary study of Comberg *et al.*[2] and while it does tend to confirm the results of the study by Comberg *et al.*, is not definitive. A longer term study is required to determine whether the hypotensive action of increased dietary linoleic acid is transitory. In addition, studies on renin and aldosterone activities would be required to clarify the mechanism involved.

SUMMARY

Concomitant with an increase in dietary linoleic acid ingestion of 1.2 percent of calories over one month, there was an eight mmHg decrease in systolic and five mm decrease in diastolic blood pressure in 28 mildly hypertensive human adults. Platelet aggregation time increased by 26 percent while disaggregation time decreased by 33 percent while on diet. Serum cholesterol decreased but not serum triglycerides. No significant changes were noted in either sodium intake or body weight while on diet. Increased dietary linoleic acid appears to be of use in lowering blood pressure in mildly hypertensive humans.

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Hallucinations

JAMES L. ELMORE, M.D., AND
ERVIN VARGA, M.D., Belle Mead*

Knowledgeable evaluation of hallucinations can make a significant contribution to diagnosis and treatment of organic and functional disorders. The clinical value of careful analysis of this perceptual disorder is emphasized. Hallucinations provide etiological clues to the understanding of regression in functional psychoses, especially schizophrenia.

Hallucinations may be the presenting symptom of an illness or may occur in delirium tremens during withdrawal from alcohol, in toxic drug states, infectious diseases, metabolic disorders, post-traumatic states, uncompensated cardiac or pulmonary failure impairing cerebral function, and in primary central nervous system disease. Several major psychiatric conditions, including psychotic depression, manic-depressive reaction, and schizophrenia (in which hallucinations are frequent) often are preceded by stress, especially serious physical disease. Hallucinations, then, are of considerable importance in the diagnosis and treatment of both major medical and psychiatric conditions.

DISCUSSION

A classification of hallucinations is worthwhile and assists in the differential diagnosis of the underlying disease, as certain types of hallucinations are characteristic of specific conditions. They can be classified as sensory distortions (illusions), sensory deceptions (hallucinations), and special types of hallucinatory experiences. Both illusions and hallucinations have all the characteristics of real perception and seem real to the individual. They are vivid and are not controlled by the will[1]

Pain Illusions—Illusions are observed in various forms.[2] There may be a change in the intensity of perception of pain, for example. Hyperesthesia, an increased sensitivity to pain and other stimuli, may occur under stress and in hypochondriacal states. Hypesthesia, or decreased sensitivity to stimuli, may occur in delirium and under any stress. A case in point is that of the soldier who may not feel pain when wounded during the heat of battle.

Visual Illusions—Changes may occur in the quality and content of visual perception. Colorvision, a phenomenon occurring under drug effect, is a feeling that one is wearing sunglasses with yellow or purple lenses. In macropsia, objects appear larger than they are. Micropsia (small vision), along with macropsia, may occur in organic brain syndrome (OBS) and in schizophrenia. A schizophrenic young man recently told the senior author that he appeared much smaller than he really was. This experience is viewed as an attempt by the individual to overcome fear of the therapist who was perceived as powerful and threatening.

Illusions or sensory distortions involve misinterpretation. An external stimulus is present but is interpreted inaccurately. Such phenomena occur frequently in normal, healthy persons under stress or in anxiety states. Illusions are quite common in children. A curtain in a window at night may be perceived as an invasive arm, thoroughly frightening the child. Organic brain syndrome often is associated with illusionary experiences such as the misidentification which

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occurs when the senile patient thinks he recognizes his mother in an approaching nurse.

Auditory Hallucinations—Auditory hallucinations are most frequent in functional or non-organic psychoses. One may hear music, speech which can be imperative, or conversations. They can be supportive, self-healing voices or one's own thoughts (echo hallucinations).

Loud, clear hallucinatory experiences are characteristic of schizophrenia and alcoholic hallucinosis. They generally occur with clear consciousness without other psychopathological signs. Frequently, two or more persons are perceived speaking critically about the patient. Derogatory hallucinations may occur in depression and in involutional psychosis as well as in schizophrenia.

Visual Hallucinations—Visual hallucinations are more frequent in organic brain syndrome, especially in acute states such as delirium tremens or drug-induced psychoses, LSD intoxications, or epileptic psychosis. Cardiac failure or pulmonary disease compromising adequate cerebral oxygenation can precipitate this type of hallucination. Visual hallucinations, although not as common as auditory hallucinations, frequently are noted in schizophrenia.

Olfactory Hallucinations—Olfactory hallucinations most often are seen in hypersensitive, neurotic persons, especially in hysterics who may smell bad odors everywhere. They occur in involutional psychosis and may be associated with the paranoid delusion of being poisoned. A burnt rubber smell is characteristic of temporal lobe tumor.

Tactile Hallucinations—Tactile hallucinations occur mostly in hysteria. They are likely to be sexually charged. For example, a hysterical patient may feel that she has been raped or molested during hypnosis. Tactile hallucinations also may occur in cocaine addicts, as well as in schizophrenics.

Vestibular Hallucinations—Vestibular hallucinations are typical in two conditions. They may be secondary to encroachment of ponto-cerebellar tumors on the vestibular apparatus of the ear or they are LSD-induced. They may be expressed as the loss of sense of gravity and a feeling of being able to fly. These are to be distinguished from depersonalization states. During periods of great emotional stress or personal threat, an individual briefly may experience a sense of depersonalization (loss of personal identity) associated with a feeling of floating weightlessly.

Unusual Hallucinations—Unusual "presence" hallucinations occur when the person does not hear or see people around him, but does feel their presence. Hysterics and schizophrenics suffer with this type of hallucination. Several other types of hallucination are known. In the so-called pseudo-hallucination, the patient does not project his hallucinations in real outer space, but may speak of voices coming from within his head or from his ear[3]. One may locate the origin of the incoming stimuli in outer space and may speak of "messages from the White House," or telecommunications from the stars. They are typical of schizophrenia. Paranoid patients may falsify their memories with resulting fabrications; for example, a woman may say, "I was married to Frank for three weeks," when, in reality, she never was. The hallucinations which sometimes occur during the altered states of consciousness while falling asleep or at the time of wakening are absolutely normal phenomena.

Organic Causes of Hallucinations—The etiology of hallucinations is multiple. We know that exogenous toxins such as lysergic acid diethylamide (LSD, "acid") and phencyclidine (PCP, "angel dust") and other psychotomimetics can produce hallucinatory experiences, as can direct cerebral injury and stimulation during brain surgery. Toxic, infectious, and metabolic disorders, and cardiac and pulmonary failure may impair cerebral functioning with secondary occurrence of hallucinations. Hallucinations, then, may be related to organic or chemical changes in the body. They also occur in the face of stresses upon the individual leading to a regression from an adult to an infantile level of adjustment, thinking, and personality functioning.

Etiology of Psychogenic Hallucinations—In psychiatric conditions, the hallucination is valuable as an effective guide to appropriate treatment. Hallucinations are related intimately to important inner motives and conflicts of the individual. They offer the physician a means of understanding the patient's experience in the early stages of treatment, when the patient may be too disturbed to participate in rational discussion.

An understanding of the genesis of hallucinations is helpful in treatment. Freud developed a theory which remains useful.[4] He held that when a hungry baby is fed and satisfied, a memory trace of this satisfaction is established in the brain. The rewarding perception of nourishment is associated with the memory trace of hunger. When hunger recurs, the baby attempts to reexperience internally the original feeling of satisfaction. The impulse to reestablish the the satisfaction is called a wish; the reappearance of the feeling in the form of a hallucination is the fulfillment of the wish.

According to Freud, hallucinations represent the emergence from the unconscious mind into the conscious mind of previously repressed images in response to the psychological needs of the individual. This generally occurs in the presence of marked anxiety feelings and may be viewed as an attempt to relieve intolerable anxiety from ungratified wishes, low self-esteem, self-criticism, or guilt, or from the tension of repressed impulses and a corresponding desire for a more satisfying reality. External reality which is not consistent with the hallucination generally is ignored. Hallucinations, then, represent a return by the patient to a level of logic and perception which, though normal for a small child, is unacceptable in the adult.

Hallucinations may take many different forms, but the form usually is related to the psychic purpose, and it is this fact which makes them a revealing source of information for the therapist. Thus, projection of unbearable guilt occurs when one hears accusing voices. Fear of unacceptable aspects of the personality may be represented by the sight of terrifying animals, and feelings of being morally bad may be expressed by the perception of foul odors.

Most people can be induced to regress to earlier levels of adjustment when enough stress and anxiety is generated. However, there are predisposing factors in the early life and development of those most prone to undergo a severe regression with the development of hallucinations and the frequently associated delusions.

FAMILY INFLUENCES

Marked psychological regression resulting in the breakdown of adult logical thinking and development of hallucinations is characteristic of schizophrenia. One recent sophisticated and well-controlled study of family interaction has identified the type of power structure, communication styles, and feeling states in families rearing schizophrenic offspring[5].

There are marked disturbances in several areas studied.

Power relationships in the family are not clear, and there is no agreement in this area among the members. There is marked fusion between father, mother, and the psychotic offspring. Interpersonal negotiation is inadequate or impossible. The family does not permit development of true autonomy and self-expression; instead, one member often speaks for another, invasively telling him what he thinks or feels. There is a concomitant failure to take responsibility for one's own feelings.

Perception of reality is disturbed; the members may hold beliefs that are totally inconsistent with reality. The family may not recognize, for example, that one member is blatantly psychotic. In addition, a distinctly negative emotional climate characterizes severely disturbed families and includes feeling states such as hostility, depression, hopelessness, and despair.

GENETIC FACTORS

Kety, reviewing genetic studies, suggests that schizophrenia may be a heterogenous syndrome with varying degrees of genetic and environmental determination[6]. Further, he notes that biochemical disturbances exist, since the genes operate through biochemical mechanisms. Thus, some of the functional or psychogenic psychoses may have an underlying, as yet poorly understood, biochemical basis for their associated hallucinations.

MANAGEMENT

The presence of hallucinations provides diagnostic information about the nature of the underlying disorder. Many physical and neurological illnesses may precipitate hallucinations; correct management involves treatment of the underlying disorder. In the absence of trauma, or physical or neurological abnormalities, one seriously may consider the onset of a psychogenic disorder. When an individual regresses to a psychotic schizophrenic adjustment with its attendant hallucinations, it is necessary to reestablish mature personality function by lowering excessive anxiety through medications and supportive therapy with the patient and family and, if necessary, with hospitalization.

SUMMARY

Major disorders, both physical and mental, may be attended by hallucinations. They are observed frequently in toxic drug states, infections and metabolic disorders, cardiac failure, and organic brain syndrome, as well as in depression, manic-depressive episodes, and schizophrenia. Their presence can assist the physician in accurate diagnosis and skillful management of the patient.

Illusions (sensory distortions) result from misinterpretation, i.e., an external stimulus is present but inaccurately interpreted. They occur in organic brain syndrome, hysteria, drug psychosis, and schizophrenia, and in anxiety states.

Each of the senses is subject to deception, and hallucinations (sensory deceptions) occur in the auditory, visual, olfactory, and tactile senses, as well as in the vestibular apparatus. They happen in the absence of external stimuli and represent the transition from thought to the perceptual sphere. Hallucinations may occur in the normal person at the time of falling asleep or awakening.

Some hallucinations have an organic etiology such as exogenous toxins and direct cerebral injury, or they may be secondary to impaired cerebral function resulting from physical illness such as delirium.

Functional or non-organically induced hallucinations are secondary to psychological regressions under stress and represent the emergence into consciousness of repressed images and impulses. They are associated with the return to a level of logic and perception found in young children. The content of the hallucination is related to its psychic purpose, e.g., projection of unbearable guilt occurs when one hears accusing voices.

Early detrimental rearing experiences impair adequacy, self-esteem, and security resulting in greater vulnerability to regression. Kety has shown, in addition, that genetic as well as environmental factors are involved, especially in certain early, severe forms of regression into schizophrenia with its hallucinatory experiences.

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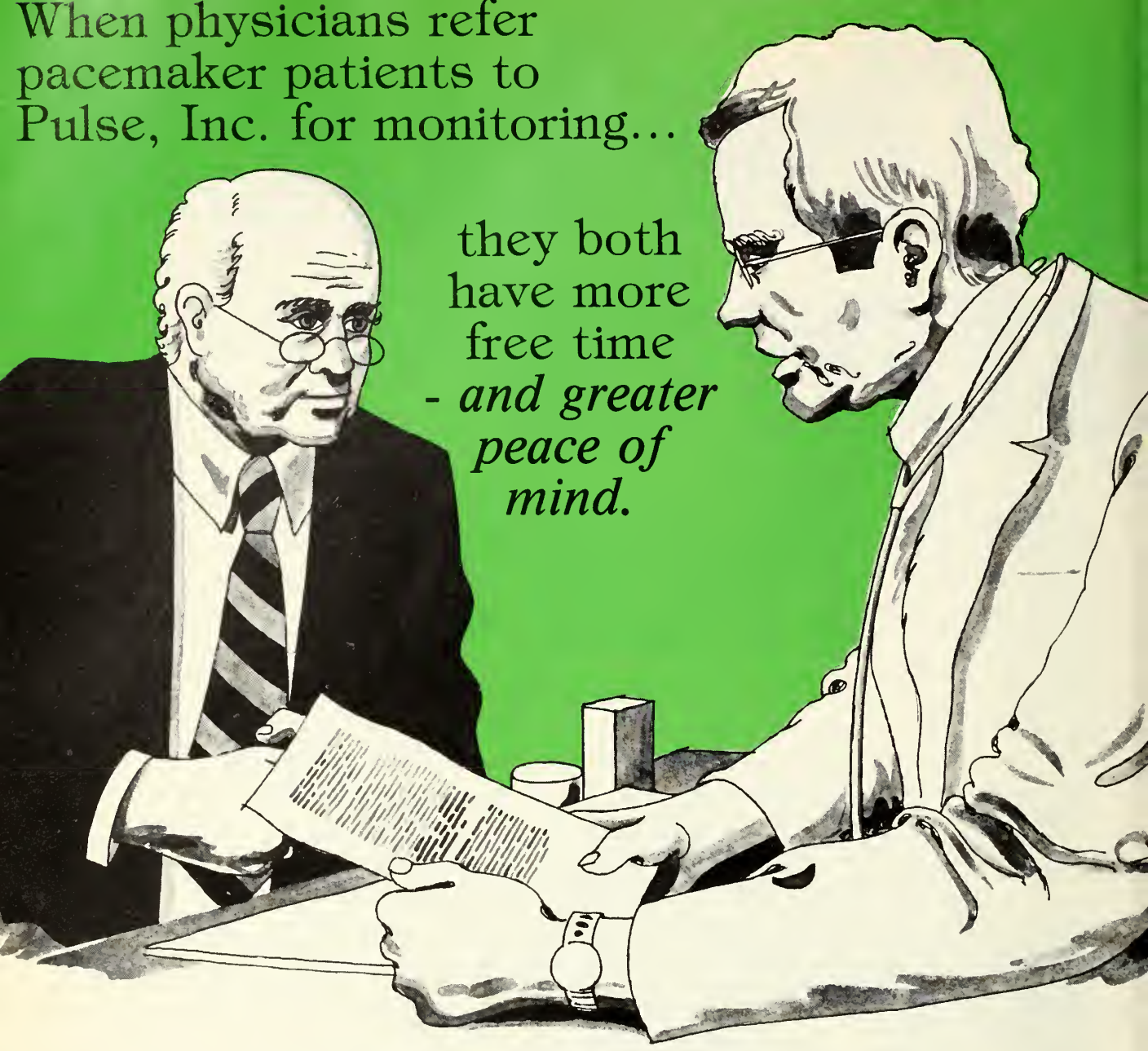
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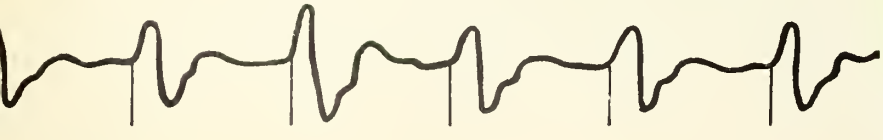
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Epistaxis, Pituitary Adenoma and Acromegaly*

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Pituitary tumors uncommonly extend into the nasal cavity, but more frequently present in the nasopharynx, presumably by direct extension through the sphenoid sinus. This paper describes a patient who had nasal symptoms resulting from a rather extensive pituitary adenoma. The tumor was hormonally active producing very high titers of human growth hormone and prolactin. Clinical data, surgical treatment, immunocytochemistry, and electron microscopy are reported. A relevant literature search is included.

Pituitary neoplasms are uncommon and are detected clinically because of locally expansile growth or an endocrinopathy resulting from abnormal hormonal activity. Various pathways of extrasellar extension have been described and most of these pathways remain intracranial. There are a number of cases reported in which the pituitary tumor has extended into the nasopharynx, but there are only eight published reports in which the tumor extended into the nasal cavity. We recently treated a patient who was acromegalic because of a large pituitary chromophobe adenoma. She initially was seen for recurrent epistaxis and nasal obstruction due to extension of the pituitary tumor into the left nasal cavity.

This case describes the first patient in whom a pituitary tumor presented as epistaxis. The clinical acromegaly is documented photographically and radiographically as well as by serial measurement of growth hormone and prolactin levels. In addition, the tissue was examined by immunocytochemical techniques and electron microscopy in an attempt to determine the cell of origin of the excessive amounts of growth hormone and prolactin.

CASE REPORT

A 42-year-old female was seen in September, 1976 at the Newark Eye and Ear Infirmary with recurrent left anterior epistaxis and unilateral nasal obstruction. Epistaxis occurred intermittently during the preceding six months and nasal obstruction had been present for approximately one year. The bleeding was seen to originate from a mass in the posterior aspect of the left nasal cavity. The mass was biopsied and anterior nasal packing was placed for control of the hemorrhage.

Additional history revealed a two-year period of vague frontal headaches with no subjective visual difficulty. The patient had noticed a disproportionate enlargement of her face, hands, and feet. Numerous family members and friends commented on the enlargement of her nose, ears and jaw, (Figures 1A and 1B). Examination of a personal photograph taken in 1970 showed that the patient's appearance had indeed changed as she described (Figure 1C). Two years earlier the patient had to have a wedding ring cut from her finger because "it became too tight." Also her shoe size had increased from nine to eleven. Gynecological history was significant in that the patient had three pregnancies with normal children and no abortions. Menses had been ir-

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Figures 1-A Anterior View 1976, 1-B Lateral View 1976, 1-C Anterior View 1970—Obvious enlargement of facial features in comparison with photograph from 1970.

regular for the preceding seven months.

Physical examination at the time of admission disclosed a rather masculine-appearing female with the clinical features of classical acromegaly. The vital signs were normal with a blood pressure of 130/70. Examination of the facial features showed furrowing of the brow and prominence of the nose, ears, and malar eminences. The nose was bulbous and the nasal skin showed increased sebaceous gland activity. There was a mass in the left nasal cavity completely obstructing the airway. The septum was in the midline and the turbinates appeared normal. Visual acuity was grossly normal and there was no ocular muscle paresis. Fundoscopic examination showed temporal pallor of both discs. Visual fields were concentrically constricted to 30 degrees. The mandible was enlarged and elongated and there was a class III malocclusion. The neck and thyroid gland were normal to palpation. The chest was normal. The heart was moderately hypertrophied by electrocardiographic criteria, but there was no irregularity of rate or rhythm. On breast examination milk was easily expressed from both nipples. With the exception of disproportionate enlargement of the hands and feet, the remainder of the physical examination was unremarkable.

Laboratory studies showed that the electrolytes, complete blood count, urinalysis, and serum and urine osmolalities were normal. All components of the SMA 12 also were normal with the exception of the serum inorganic phosphorus which was 6.2 mg/dl (N=2.5-4.5). Coagulation studies were within the normal range. A five-hour glucose tolerance test did not demonstrate chemical diabetes. Twenty-four hour urinary 17 hydroxy and 17 ketosteroids were normal. Serum Thyroxin (T4) was 14.2 μg/dl (N=4-12), triiodothyronine uptake (T3U) = 1.37 (N=0.83-1.7), free

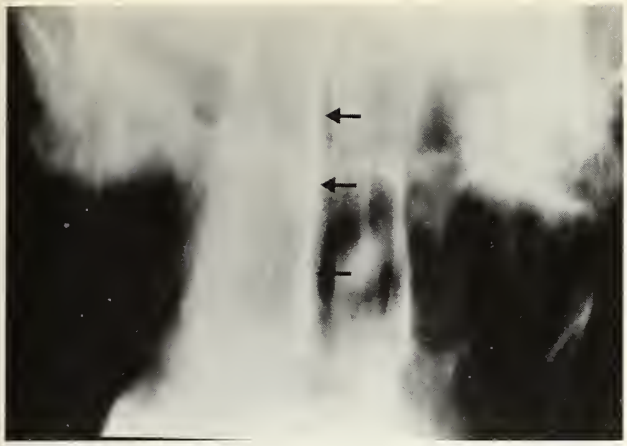


Figure 2—AP Laminogram of sinuses demonstrating opacification of left ethmoid and sphenoid sinuses and left nasal cavity with evidence of bone destruction.



Figure 3—Lateral skull radiograph showing markedly enlarged sella turcica with loss of anterior and posterior clinoid processes.

thyroxine index = 19.5 μg/dl (N=4-12). Growth hormone assays were performed in conjunction with the glucose tolerance test and the results are as follows: Random specimen = 680 ng/ml (N=0-6 ng/ml), fasting = 433 ng/ml, 1/2 hour = 588 ng/ml, 1 hour = 611 ng/ml, 2 hours = 513 ng/ml, 3 hours = 623 ng/ml. Thyroid stimulating hormone (TSH) was 5.85 mu/ml (N=2.0-11.0). Prolactin was 38.6 ng/ml (N=0-3). Estradiol level was less than 12 picogm/ml (normal). Plasma cortisol, FSH, and LH were normal.

X-ray studies of the paranasal sinuses by plain films and tomography demonstrated opacification of the left ethmoid and sphenoid sinuses and the left nasal cavity by a soft tissue density (Figure 2). Skull x-rays showed hyperostosis frontalis interna and a markedly enlarged sella turcica which measured greater than 20 mm in diameter (Figure 3). Sellar tomograms demonstrated destruction of the left anterior clinoid process and thinning of the posterior clinoids as well. There was erosion of the floor of the pituitary fossa and destruction of the dorsum sellae. A soft tissue mass filled the sphenoid sinuses. X-rays of the hands and feet (Figure 4) demonstrated a positive "Sesamoid Sign" and tufting of the terminal phalangies, both of which are radiologic signs of acromegaly. Selective right and left carotid arteriograms showed no evidence of suprasellar extension of the pituitary tumor. Computerized axial tomography demonstrated a mass lesion in the region of the pituitary fossa with erosion of the anterior clinoids, (Figure 5).

Tissue from the nasal biopsy was consistent with pituitary adenoma. With a clinical diagnosis of acromegaly, laboratory documentation of a markedly elevated growth hormone level, and biopsy evidence of an intranasal pituitary tumor, surgery was performed on October 28, 1976. Through a sublabial incision the nasal septum was removed and a transsphenoidal hypophysectomy performed. As the region of the sphenoid sinus was approached, it became apparent that the anterior walls and floor of the sinus were absent. The anterior wall of the sella turcica also was absent. No large tumor was identified but the tumor infiltrated along the mucosa of the sphenoid sinus extending directly onto the nasal septum. All gross tumor and abnormal appearing mucosa were removed. Tumor also extended onto and seemed to invade the underlying dura so a small portion of dura was removed as well. It was felt to be impossible to remove all involved dura so it seemed certain that some pathologic pituitary tissue was left behind. A fascia lata-muscle graft was used to obliterate the sphenoid sinus and was held in place with nasal packing impregnated with antibiotic ointment.

Postoperatively, the patient was kept at bedrest and placed on intravenous cephalasporin. Intake and output were carefully recorded. On the first postoperative day she began to experience intense thirst. Her oral intake of fluids increased as did her urine output, which over the next two weeks ranged from 1000 to 8000 cc per 24 hours. Although urinary and serum electrolytes and osmolalities were checked repeatedly, a diagnosis of true diabetes insipidus could not be made. Oral intake and urinary output eventually equilibrated and fluid balance did not develop into a significant problem. There was a postoperative temperature elevation which was "spiking" in nature and ranged from 37 to 40.6 degrees C. This was associated with constant frontal and midfacial pain. On the seventh postoperative day, seven yards of packing were removed from the nasal cavity and she remained afebrile from this point until the time of discharge.

On the eleventh postoperative day (four days after removal of nasal packing), the patient began to complain of a watery nasal discharge. Cerebrospinal fluid (CSF) rhinorrhea was documented and prophylactic gentamycin and carbenicillin were given. Over the next ten days the CSF rhinorrhea gradually decreased and eventually ceased.

By the twenty-sixth postoperative day the patient was eating a regular diet and was free of complaints. When discharged her medication included thyroid and glucocorticoid hormone replacement.

Two months after hospital discharge serum hormone levels were again tested. Thyroid function was normal. Serum growth hormone level was down to 32 ngm/ml from 680 and prolactin was 10.1 ngm/ml compared to 38.6 preoperatively. These elevated results indicate persistence of the presence of functioning tumor producing both growth hormone and prolactin.

At the time of this writing the patient is being evaluated as a possible candidate for postoperative radiotherapy to control the acromegaly.

PATHOLOGY

Histology (Figure 6)—Biopsy of the nasal mass showed a diffuse growth of cuboidal cells with round centrally placed regular nuclei and moderate cytoplasm. The stromal element was predominantly vascular and rather scant. Mallory's aniline blue for pituitary granules showed mainly agranular chromophobe elements with scattered sparsely granular



Figure 4—Radiograph of right foot showing prominent sesamoid bones and tufting of the terminal phalanges.

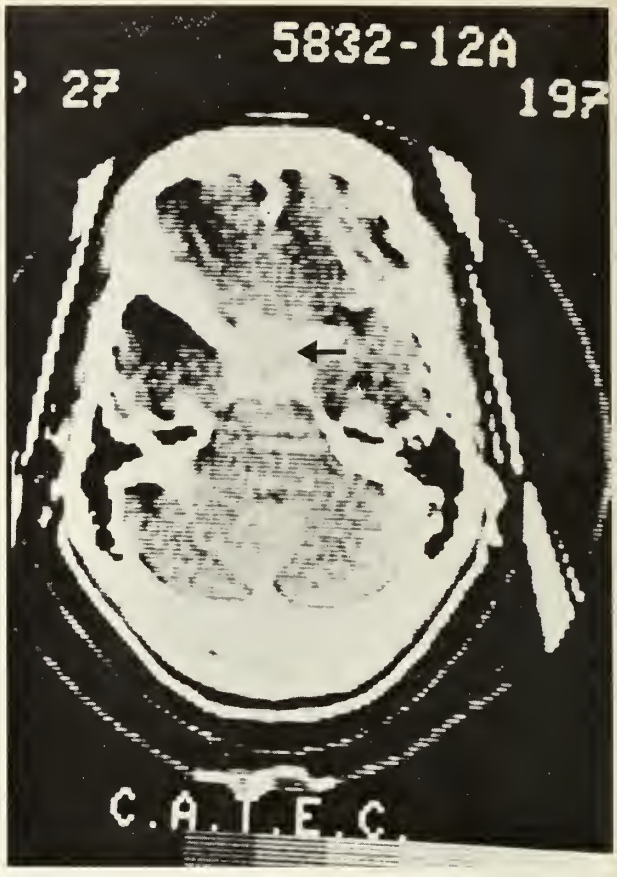


Figure 5—Computerized tomogram with contrast showing a large mass lesion in the region of the pituitary fossa with erosion of the anterior clinoids.

eosinophils. An occasional cell contained dense eosinophilic granules. The excised tumor showed identical morphology, but it contained foci of bone involvement. The fragments of excised adenoma did not have a capsule. Mitotic activity was present, but not prominent.

Immuno-Cytochemistry[1,2]—Five micron sections of the biopsy material were deparaffinized and layered with anti-human growth hormone and incubated for one hour. This was followed by anti-sheep antibody of rabbit origin and subsequent treatment with anti-rabbit, antiserum-horse radish peroxidase conjugate. The peroxidase then was stained with benzidine according to Karnovsky. Similar procedure was repeated with antibovine anti-prolactin. Rhesus and human pituitary controls were used to check the anti-sera. Antibody against growth hormone was found to react with many of the tumor cells while the anti-prolactin was found to react with an occasional cell. Since the latter cells were so sparse, one could not determine whether the same cells reacted with both anti-growth and anti-prolactin substances.

Electron Microscopy[3] (Figure 7)—Some fragments of the excised tumor tissue were fixed in two percent gluteraldehyde as soon after removal as possible. This was then embedded in plastic, sectioned, and examined under an electron microscope. The cells contained poorly preserved endoplasmic reticulin, many swollen mitochondria, rare lipid bodies, and small secretory granules. These granules did not vary much in size and were scant in number. They were mainly located at the periphery of the cell. A two-cell population was not discernible. This may be due to either a one-cell population or the fact that in our sample of tissue the "lactrophs" were few in number. Thus, electron microscopic evidence for or against a one or two-cell population is not present. However, the immuno-histochemistry is suggestive of a two-cell adenoma. The predominant cell is the growth hormone secreting cell with few scattered lactotrophs secreting prolactin.

DISCUSSION

Pituitary tumors are not uncommon; they comprise approximately 10 percent of all intracranial neoplasms.[4] They generally are benign adenomas, most often situated within the sella turcica. They usually are of clinical significance because of an endocrinopathy resulting from functioning hormonal activity or because of encroachment on neighboring structures due to local expansile growth of the tumor.

Not uncommonly pituitary tumors can escape the boundaries of the sella turcica as they increase in size. Jefferson discussed in detail the factors responsible for extracellar extension of pituitary tumors.[5] He considered six possible pathways of growth outside of the usual boundaries of the pituitary fossa. His findings were reviewed by White and Warren.[6] The most common pathway is by pharyngeal extension through the bony floor of the sella which allows the tumor to expand into the sphenoid sinus. From here it may extend into the ethmoid sinuses or into the nasopharynx. A second pathway of extrasellar growth is *via* hypothalamic extension in which the tumor projects upward into the third ventricle behind the optic chiasm. With temporal extension the tumor projects laterally between the chiasm and the cavernous sinus and may continue to grow into the middle cranial fossa. A fourth pathway involves direct invasion of the cavernous sinus and further lateral

growth under the dura of the floor of the middle fossa into the trigeminal root sheath (Meckel's Cave). The fifth pathway is posterior subtentorial extension in the region of the cerebellopontine angle. The rarest mode of extrasellar extension would appear to be in the frontal direction; here the tumor extends along the floor of the anterior cranial fossa separating and compressing the frontal lobes.

As mentioned above, the most common pathway of extracellar extension is in the inferior direction into the sphenoid sinus. In spite of this fact there are not many reports of direct extension into the nasopharynx and there are even fewer reports of extension into the nasal cavity. Henderson, in 1939, reviewed 338 cases of Dr. Harvey Cushing's original series and found nasopharyngeal extension in only eight cases.[7] He found no cases which involved the nasal cavity. In fact, in Cushing's original monograph in 1932, he made no mention of intranasal extension of pituitary tumors.[8] Since that time there have been reported only eight cases of direct intranasal extension of a pituitary tumor originating in the sella turcica in the English language literature.

Bailey and Cutter reported the first case of a chromophobe adenoma which invaded the nasal cavity in 1940.[9] The patient presented with obstruction by a nasal polyp which histologically proved to be a pituitary adenoma. At autopsy the tumor was in direct continuity with the pituitary fossa. The second reported case of a nasal pituitary tumor was by Shea in 1941.[10] The tumor filled the vestibule of each naris. There was radiologic evidence of obliteration of the sella and extensive destruction of the floor of the middle cranial fossa.

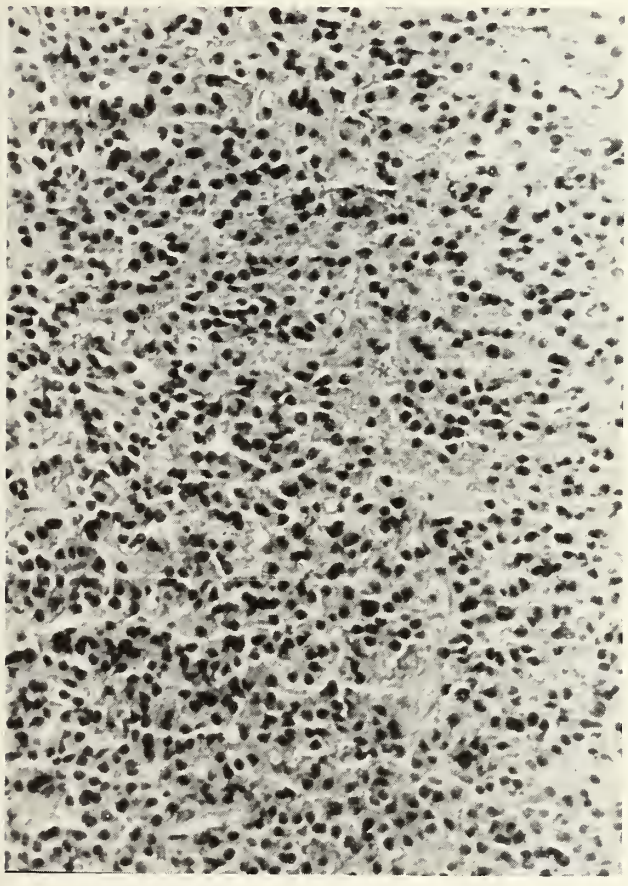


Figure 6—H & E of pituitary adenoma showing groups and nests of small cells with uniformly staining cytoplasm and round nuclei of even size. There is a vascular stroma, and the nests of cells are separated by thin fibrous septa.

In 1950, Kay reported three additional cases of chromophobe adenomas which involved the nasal cavity.[11] All three cases were encountered during one year's time at the Columbia-Presbyterian Medical Center in New York City. The first patient presented with facial pain, headache, changes in sensorium, anisocoria, strabismus, and paresis of the superior oblique muscle. He had a large suprasellar lesion and polyps in the left posterior ethmoidal region which proved to be pituitary adenoma. The second patient presented with bitemporal hemianopsia. Polyps of pituitary tissue were found in both middle meati. The third patient presented with progressive loss of vision in one eye and ipsilateral frontal headache. He had a deformed sella and a nasopharyngeal polyp which originated in the ipsilateral posterior ethmoidal region. Histologically it was a pituitary adenoma.

The sixth case, reported by Sheline in 1964, was in a patient with an enlarged sella turcica and a tumor extending inferiorly and attaching to the posterior nasal septum.[13] In 1974, Sadick reported a case of an individual with acromegaly who presented with bilateral nasal obstruction and a large suprasellar tumor.[12] Polypoid masses of pituitary tissue filled both nasal cavities and their vestibules. More recently, in 1976, Summers reported a man who had received a full course of radiation therapy 21 years previously for a suspected pituitary tumor based on visual field defects.[14] He presented with bilateral nasal obstruction from tissue which was confirmed to be chromophobe adenoma.

There are some reports of nasopharyngeal pituitary tumors arising from ectopic pharyngeal pituitary tissue. These tumors show no continuity with the sella turcica and the intrasellar pituitary gland is apparently normal in size and function. For further discussion of this "pharyngeal pituitary gland" the reader is referred elsewhere.[15-17]

Extension of a pituitary tumor into the nasal cavity obviously is not a common occurrence. We add our case to the short list of cases reported in the medical literature. Our patient presented with classical acromegaly but actually sought medical attention only because of recurrent nasal bleeding. She is the first reported patient to present with epistaxis due to intranasal extension of a pituitary tumor. It is also of interest to note the degree of hormonal activity of this tumor. A random growth hormone level measured preoperatively was 680 ng/ml. Endocrinologists who treat acromegaly with some frequency usually report growth hormone levels in the range of 100-150 ng/ml.

The presence of markedly elevated levels of serum growth hormone and prolactin prompted us to make a histochemical search for two separate cell types. Both biopsy and surgical specimens were examined by immunocytochemical techniques and electron microscopy in an attempt to identify the cells of origin of the two hormones. Two distinct cell populations were identified. The results of testing with anti-human growth hormone and anti-prolactin suggested a two cell population: the predominant cell, a somatotroph, producing somatotrophic hormone, and a much less abundant cell, the lactotroph, secreting prolactin. Electron microscopy neither confirmed nor disproved this finding.

SUMMARY

This article reviews the English language literature pertaining to the extension of pituitary tumors into the nasal cavity. We present a patient with obvious acromegaly due to a large pituitary adenoma. She sought medical attention because of recurrent nosebleeding. This case brings the

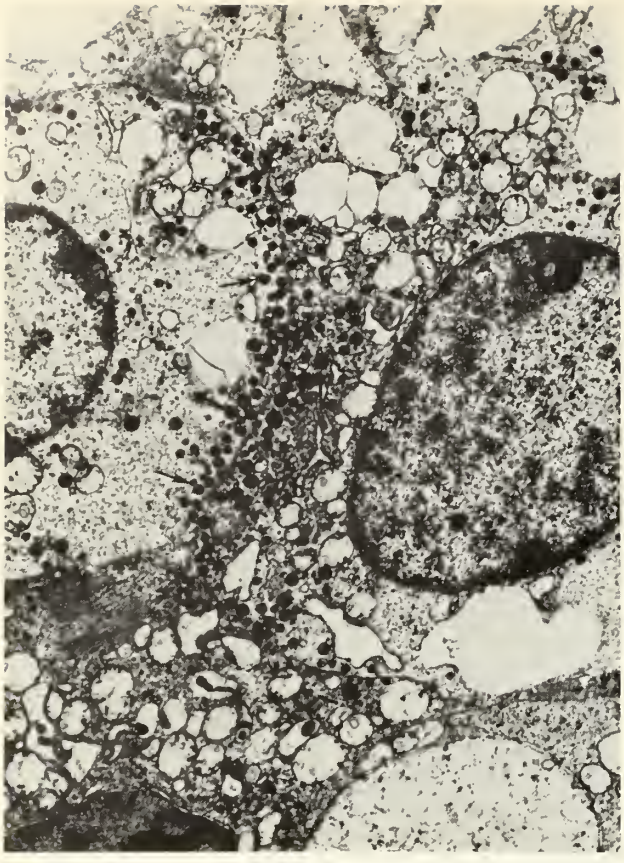


Figure 7—Electron micrograph shows secretory granules within the adenoma cells.

reported total to nine. The history, physical findings, and hospital course, including photographic and radiographic documentation are discussed. Immunocytochemical and electron-microscopic studies are included in an attempt to identify the cells of origin of the hormonally active substances.

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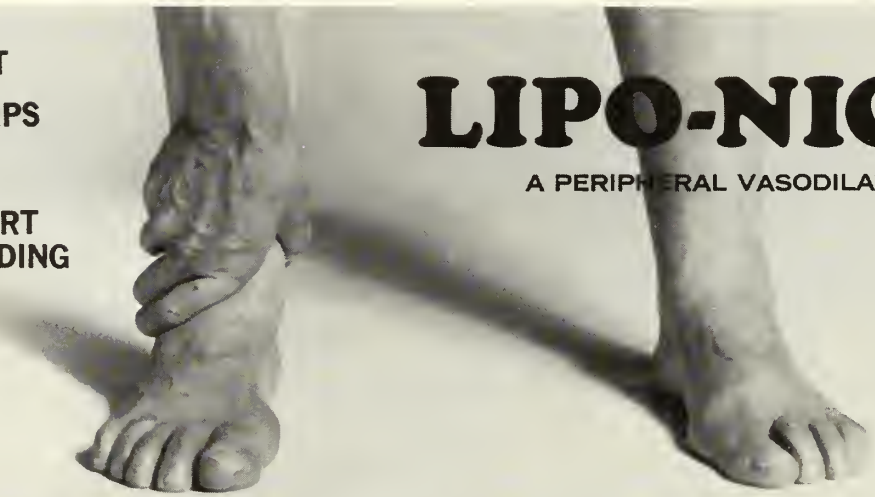
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Emergency Use of Cimetidine in Acute Gastrointestinal Hemorrhage: Endoscopic Evaluation*

JEROME H. SIEGEL, M.D.,
FRANK M. CANDIDO, M.D., and
WILLIAM S. ROSENTHAL, M.D., Emerson

The efficacy of cimetidine in the treatment of acute duodenal ulcer has not been established. The following case report describes recurrent bleeding from a duodenal ulcer which had been plicated surgically. The bleeding ceased after intravenous administration of cimetidine. Healing of the lesion occurred in three weeks and was documented endoscopically.

Cimetidine, an H_2 receptor antihistamine antagonist, has been shown to inhibit gastric acid secretion in normals and patients with duodenal ulcer.[1-7] Blockade of acid secretion occurs not only with histamine stimulation but also with gastrin, caffeine, and cholinergic-stimulated acid secretion.[5,8,9]

No reports concerning the use of the H_2 receptor antagonist cimetidine in gastrointestinal bleeding were available when therapy was initiated in the case described below. Treatment of this patient was instituted in November 1976 when only one report of treatment of stress-induced gastrointestinal bleeding with metiamide was recorded.[10] Ongoing studies using cimetidine now are entered into the literature.[11,12] The case described herein documents the effectiveness of cimetidine in intractable gastrointestinal bleeding.

CASE REPORT

A 61-year-old alcoholic Puerto Rican male was hospitalized in September 1976 with acute upper gastrointestinal hemorrhage from bleeding gastric varices. This problem responded to medical management which included blood replacement. Later, hepatocellular failure was documented by the development of ascites, jaundice, and an elevated prothrombin time. Laboratory data did not disclose azotemia or electrolyte abnormality. The ascites responded to spironolactone diuresis. Mild hepatic encephalopathy was treated successfully with neomycin, and later, with lactulose. Over a four-week period a slow but gradual improvement occured.

Transhepatic percutaneous angiographic studies demonstrated a patent portal vein and portal vein pressure of 33 cm of H_2O. Extensive collateral circulation was demonstrated and, in addition to the gastric varices, a large inferior mesenteric collateral venous circulation was identified. The patient was prepared for and underwent splenorenal shunt which was performed successfully. The immediate postoperative course was marked by disappearance of ascites. There was a progressive rise in the serum bilirubin and serum albumin and protein decreased. Mild encephalopathy recurred and was treated successfully with lactulose.

Seven days following the portocaval shunt a severe upper gastrointestinal hemorrhage requiring infusion of 12 units of blood recurred. Angiography was performed at that time and demonstrated a patent shunt and a collection of contrast material in the area of the duodenal bulb. An emergency laparotomy was performed and a pyloroplasty and plication of the acute duodenal ulcer was carried out. Once again the patient's hemoglobin and hematocrit rose following transfusions. The liver function studies remained stable and he improved. Three weeks after the pyloroplasty and ulcer

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Table 1

Events:	September 1976 Initial bleed 10 unit transfusion		October 1976 Portocovol shunt			November 1976 Bleed – 12 unit transfusion ulcer plicated		Bleed – 5 unit transfusion		Cimetidine 300 mg every 4 hours, then every 6 hours
Hgb Gms/dl	11.3		9.6			5.4		10.9		12.0
H'crit %	39%		30.6			17		31		34.4
Platelets / cumm	120,000									
Protein Gms/dl	6.9	6.0		6.3	4.6	5.8			5.6	
Albumin Gms/dl	2.4	3.3		2.9	2.1	2.5		3.1	2.8	3.0
Bilirubin mg/dl	0.9	11.6	5.2	2.8	4.3	9.4	21.4	27.6		13.4
SGOT Iu/ml	85	88	67	86	50	62		90	120	
LDH Iu/ml	160	295	320	220	385	270		250	220	206
Alkaline phosphotase Iu/ml	108	85	125	93	80	67		137	92	135
Prothrombin Time pt./control	14.6/12.9		14.3/12.4		13.0/12.9	16/12.3		14.4/11.9		
Cholesterol mg/dl	120				130			180	165	
Calcium mg/dl	8.5							8.7	8.6	
Phosphorous mg/dl	3.4								3.1	
Bun mg/dl	13		23	16	24			32		18

plication, he experienced a third episode of massive upper gastrointestinal bleeding which required an additional five units of blood. Endoscopy then demonstrated severe erosive gastritis and a large duodenal ulcer which was located in the anterior-superior portion of the duodenal bulb (Figure 1). Silk suture material was located within the ulcer bed itself and a continuous oozing of blood was observed from that area.

The patient was begun on intravenous cimetidine after emergency institutional approval and informed consent. He was given 300 mgs every four hours for 48 hours followed by 300 mgs every six hours thereafter. Following the initial dose of cimetidine, the nasogastric aspirate changed from bloody to clear. Again the patient's clinical condition stabilized. Three weeks later, while still on cimetidine therapy, the patient underwent endoscopic reevaluation. This study demonstrated a marked improvement in the erosive gastritis with the mucosa appearing less friable and erythematous. The ulcer crater demonstrated healing and no bleeding was noted. The size of the ulcer had decreased significantly (Figure 2). The cimetidine therapy was discontinued after three weeks, and the patient was discharged shortly thereafter with no recurrence of bleeding. Acid secretory studies were not performed prior to the administration of cimetidine, however, pentagastrin stimulation two weeks after discharge from the hospital demonstrated normal acid secretory function (14 meq/hr).

DISCUSSION

The mechanism of action of the histamine H_2 antagonists is that of specific receptor blockade which effectively inhibits not only histamine-mediated gastric acid secretion induced by natural physiologic stimuli[7,8,9] but also stimulation by gastrin, cholinergic agents, insulin, and food.[6,13,14] In the case presented, stress associated with portocaval shunt surgery was responsible for the development of an acute duodenal ulcer resulting in massive upper gastrointestinal bleeding. Initially this was treated surgically after failure of medical management. Despite definitive treatment, bleeding recurred.

As the patient recently had undergone surgical intervention for bleeding and was considered a poor surgical risk for a third operation, a trial by the H_2 antagonist, cimetidine, was instituted. At the time of this clinical trial, there were no published reports of its use in acute gastrointestinal bleeding.

Metiamide, an earlier H_2 receptor antagonist, had been used by MacDonald and colleagues in 11 hemodialysis patients who presented with bleeding from gastritis and/or duodenitis.[10] Their results were encouraging as bleeding was controlled effectively in nine patients. Ulcerations eroding into vascular structures and producing uncontrollable bleeding developed in the remaining two patients. In his study with an oral preparation of metiamide MacDonald postulated that the drug exerted both a local and systemic effect. This concept, however, has not been proved, and it appears more likely that the inhibitory effect of gastric acid secretion is mediated systemically.

In the case presented, the temporal relationship of the cessation of bleeding following the institution of intravenous cimetidine is regarded as substantial evidence that cimetidine was directly responsible for this effect. Cimetidine also is credited for the progressive healing of this patient's ulcer and the associated erosive gastritis, and these specific effects are attributable to the inhibition of gastric acid secretion. The favorable results experienced in this case are encouraging and the pharmacologic approach to acute or recurrent gastrointestinal bleeding in the patient at risk for surgery is certainly appealing. Further studies evaluating the effectiveness of H_2 antagonists in upper gastrointestinal bleeding are presently in progress.[11,12]

SUMMARY

Cimetidine has been shown to be effective as an H_2 receptor antagonist in inhibiting acid production mediated through physiologic stimuli such as histamine, gastrin, caffeine, and cholinergic agents. Its role in the effective treatment of peptic ulcer disease has been proved, however, there are few reports of its use in the treatment of bleeding from acute duodenal ulcer disease. This case report describes recurrent bleeding episodes in a 61-year-old male alcoholic with portal hypertension. Bleeding, occurring from an acute duodenal ulcer, following a portocaval shunt recurred despite surgical plication. The localization of the bleeding was documented by endoscopy. Following the intravenous administration of cimetidine, the bleeding stopped. Healing of the ulcer was proved by endoscopic evaluation three weeks



Figure 1—Acute duodenal ulcer outlined. Ulcer in lower area; suture from plication seen in upper portion, adjacent to mound of erythematous tissue (arrow).



Figure 1—Healing ulcer demonstrated. Mound of tissue seen is previous area where suture had been placed (arrow). Ulcer crater (healing) is outlined.

later. Cimetidine is useful in the treatment of duodenitis and peptic ulcer disease and is recommended in these conditions when associated with stress. Its use in the treatment of bleeding in peptic ulcer disease will require further investigation.

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Ultrasound Evaluation of a Spermatic Cord Myxoliposarcoma*

LAWRENCE GOULD, M.D.,
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A large myxoliposarcoma of the spermatic cord is described with the emphasis on its gray scale ultrasonographic appearance. This 1600 gram, 18 x 13 x 12 cm. mass imaged as a non-homogeneous lesion and contained echoes of variable intensity. Several quite prominent echoes were suspicious for lipid material. A secondary hydrocele was produced. Gray scale ultrasonography should be added routinely to the armamentarium used in the diagnostic evaluation of scrotal masses.

Neoplastic masses of the scrotum can be divided conveniently into those that are of testicular origin and those which are extratesticular. The extratesticular category includes tumors of the spermatic cord, the epididymis, and the testicular tunics. Tumors of the cord make up the vast majority of these growths and it is estimated that about seventy percent of them are benign.[1] Lipoma is the most common entity in the benign group. Under the malignant heading, fibrosarcoma is the most frequently encountered tumor and liposarcoma ranks second.

Recently, the fifteenth reported case of a liposarcoma was described and, in the article, the world literature was reviewed.[2] The emphasis in the report was on prognostic and therapeutic considerations.

The purpose of this communication is to present an additional case and to illustrate how gray scale ultrasonography furnished helpful information in the preoperative diagnosis.

CASE REPORT

A seventy-year-old male presented at the emergency room of the Martland Hospital Medical Center in February, 1978, with a chief complaint of a scrotal mass which had persisted for two years. Physical examination revealed a large scrotal mass which measured 16 x 12 x 10 cm. The mass was firm, non-tender, and essentially painless except for some mild discomfort. There was no edema of the skin. When the patient stood in the erect position, the weight of the scrotum was obviously heavy in the examining hand. It was impossible by ordinary manual palpation to differentiate the testis from the spermatic cord.

Auscultation of the scrotum failed to yield any bowel sound. A transillumination test for hydrocele detection was negative. A plain x-ray film of the scrotum showed solid tissue without evidence of bowel gas or internal calcification (Figure 1). An intravenous urogram and chest x-ray were normal. All laboratory studies, including HCG determinations produced normal values.

Clinically it was felt that the patient had a large testicular tumor.

Ultrasonic evaluation of the mass was done using a 2.25 MHz transducer and a commercially available gray scale machine. The entire mass was scanned from its right lateral periphery to the left lateral periphery and from its most cephalad to the most caudad limits. Transverse (Figure 2) and longitudinal (Figure 3) images were taken using a single sweep technique. A high-view gain was obtained in the longitudinal projection. (Figure 4)

A large, clearly delineated, non-homogeneous lesion with echoes of variable intensities was displayed. Areas of lobulation were noted. The testis could not be seen as a separate

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Figure 1—A plain film of the scrotum showing the large soft tissue mass.
S=Scrotum



Figure 2—A transverse echogram taken nine cm. above its most inferior border showing extent of tumor and cystic hydrocele.
R=Right
L=Left
M=Midline of anterior surface
T=Tumor (showing extent)
C=Cystic area

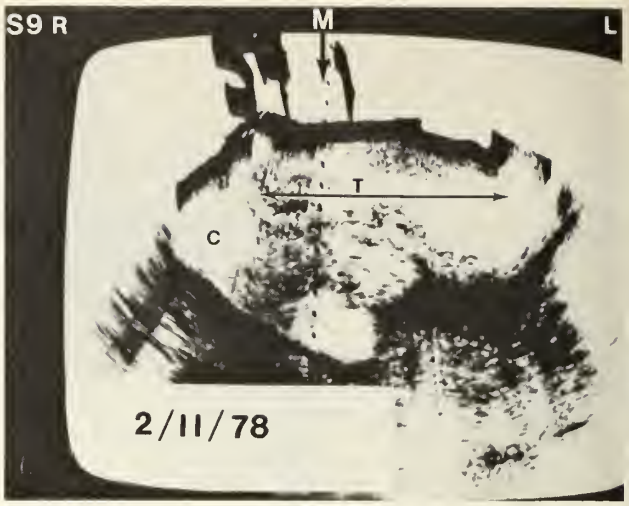


Figure 3—A longitudinal echogram taken four cm. to the right of midline demonstrating the large tumor.
H=Toward head
F=Toward feet
T=Tumor (Showing extent)
S=Caudad border of the scrotum



Figure 4—Same longitudinal echogram taken with the high gain.
H=Toward head
F=Toward feet
T=Tumor (Showing extent)
S=Caudad border of the scrotum

entity in the whole conglomerate. On the transverse cuts, a cystic area at the right periphery with a sharply defined posterior wall could be demonstrated.

It was concluded from the ultrasound study that a large lobulated neoplastic process was present either involving the cord alone or the cord in combination with the testis, but probably not the testis alone. The very high intensity echoes were quite suspicious for the presence of fat. The cystic area seen on the transverse cut was considered to be a secondary hydrocele produced by mechanical obstruction.

The patient underwent surgery and the entire contents of the right scrotum were removed en bloc.

The surgical specimen weighed 1600 grams and measured 18 x 13 x 12 cm (Figure 5). It was lobulated and rubbery in consistency. A small cystic structure could be identified on the right side which yielded about 20 cc. of clear fluid when opened. A four cm. length of spermatic cord, which adhered to the mass was seen. Gross sections revealed a gray-white glistening material with foci of yellow-fat areas and small zones of hemorrhage. An atrophic but otherwise intact testis could be identified.

Microscopic examination showed a predominant myxoid stroma in which were scattered many pleomorphic cells (Figure 6). Some cells contained lipid material which stained positively with Oil-Red-O. A histologic diagnosis of myxoliposarcoma was established. No tumor was noted either at the resected margin of the spermatic cord or in the lymphatics of the specimen.

DISCUSSION

Physical examination of the scrotum should be an easy procedure in a majority of patients. This is so because the testis is covered by only a thin layer of skin and is thus readily palpable. Other modalities used in diagnosis include



Figure 5—Myxoliposarcoma of the spermatic cord. Resected gross specimen opened to show its lobulated and glistened appearance. The hydrocele sac has been opened. The atrophic testis is not apparent in this cut.
H = Hydrocele.

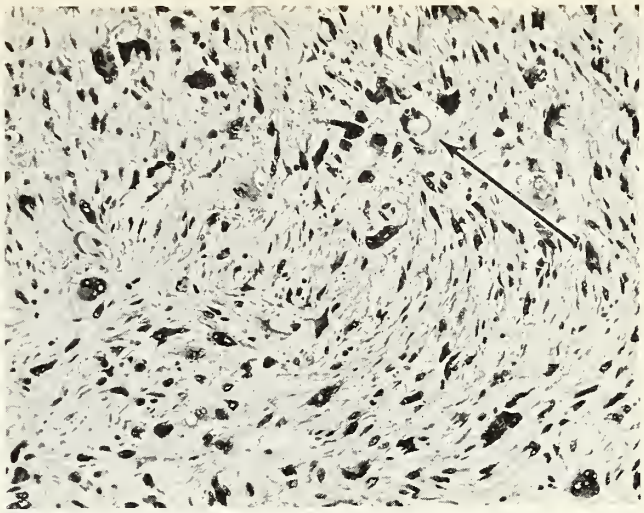


Figure 6—Photomicrograph of a representative area to demonstrate the sarcomatous cells in a loose myxoid stroma. Note the presence of a typical lipoblast cell. (arrow)
(hematoxylin and eosin, x80).

transillumination, auscultation, and soft-tissue x-rays. However, in the absence of specific pathology, (i.e., hydrocele and hernia) the efficacy of these tests is quite limited. In the presence of a huge scrotal mass, when it is important to be able to distinguish testis from cord from epididymis, a more specific method is requisite. A recent report claimed that a clear distinction could be made between the extra-testicular or the testicular origin of eighty percent of a series of chronic scrotal masses utilizing gray scale ultrasound.[3] The authors reported a characteristic homogeneous texture of the normal testis and a characteristic coarser pattern of the epididymis in their case material. The same authors also stated that they could not describe any distinguishing gray scale features for solid testicular neoplastic processes (either primary or secondary) or for those twenty percent of abnormal scrotums where both extratesticular and testicular elements were involved.

Earlier descriptions of scrotal ultrasonography are insufficient and no criteria of tumor patterns yet has emerged.[4,5]

In our case, the testis could not be separated clearly as an entity unto itself and it was our impression that this mass lesion was either a tumor of the cord alone or a growth extending from the cord into the testis destroying the anatomical distinction between the two.

The additional observation of some quite prominent or "loud" echoes in the picture led to a suspicion of the likely presence of lipid material in the mass. It is believed that fatty tissue is a network of acoustic impedance mismatches which give rise to numerous "loud" echoes. Fat also is felt to generate considerable internal reverberations.[6]

As ultrasound is an excellent method of differentiating solid from cystic structures, and as newer digital gray scale machines come onto the market with the ability to assign gray values to specific tissue densities, a catalogue of patterns of individual scrotal entities undoubtedly will unfold in the near future.

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Historical Note

Dr. Blane's Prescription

FRED B. ROGERS, M.D., Trenton*

The home of our 68th MSNJ President remains, and has been converted to good use almost a century and a half after Dr. Blane moved in. His "prescription" also lives on despite the departure of our medical forefather.

Dr. John O. Blane (1802-1885), who served as 68th president of The Medical Society of New Jersey during 1861-62, is recalled chiefly for compiling an interesting medical history of Hunterdon County. Co-authored with Drs. Samuel Lilly of Lambertville and Henry B. Nightingale of Flemington, it was published in the Society's *Transactions* for 1872. Dr. Blane earlier had been president of the Hunterdon County Medical Society (1848-49).

Today, Dr. Blane's home and office serve a pleasant function as a restaurant[1] at rural Perryville in picturesque upper Hunterdon County (New Jersey). It is located on Route 22 between Clinton and Phillipsburg.

The Blane House, where the doctor lived from 1831 until his death fifty-four years later, now is the main section of the restaurant. It was, however, known as home for other residents before Dr. Blane moved in. Erected during the colonial period of our nation, it boasts great hand-hewn timbers, thick stone walls, and large rooms with handsome fireplaces. Slate air ducts were built in for ventilation.

Dr. Blane added two wings to the house: an east wing, constructed in 1843, served as his office; the west wing, built in 1857, was a conservatory where he raised orange and lemon trees. Several years ago, a portion of the west wing was ripped out to make room for a window. A wine bottle was discovered in the masonry containing a note written by the doctor. That note is on display at the restaurant.

Born and reared in Middlesex County, John Blane received his M.D. degree from the Rutgers Medical College in 1827. A skilled physician, he tended patients in the countryside on horseback and later by wagon. Several apprentices who worked with him at Perryville became able practitioners.

The restaurant's menu today invites the diner to enjoy a sour drink called "Dr. Blane's Prescription" and to toast Dr. Blane's memory with a potion that he might have prescribed.

*Dr. Rogers may be addressed at 301 West State Street, Trenton, N.J. 08618.

[1]Coach N' Paddock

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SPECIAL ARTICLE

ECHO—New Jersey's First Operational IPA

BERNARD ROBINS, M.D., Springfield*

Since 1883 when the Mayo Clinic was established as the first group practice in the United States, the number of groups has increased until in 1969 the AMA reported 6,371. In 1929, the Farmer's Union-Cooperative Health Association in Oklahoma became the first group practice to introduce prepayment for its members. In Los Angeles the physician-owned Ross-Loos Medical Group, which is the nation's oldest continually functioning group practice health maintenance organization (HMO), also began to offer prepaid health services to employee groups in 1929. From these innovations has emerged the concept of the health maintenance organization. Within a pluralistic health care system the HMO offers an alternative manner of organizing, delivering, and financing health care services.

The largest group practice type of HMO is the Kaiser-Permanente Medical Care Program; established in 1933, it now operates in six states and serves nearly three million members. Private insurance companies also have sponsored group practice HMOs and Blue Cross and Blue Shield have become involved in developing some group practice HMOs. With the HMO Act of 1973, the federal government committed substantial funds to stimulate the development of HMOs throughout the country. The Federal Government then mandated that employers of 25 or more workers who provided health care must offer HMO coverage to employees in those areas where qualified HMOs are operating. While government support has increased the availability of HMO memberships, the precipitous rise in providing health benefits has prompted some major employers like R.J. Reynolds Industries and Ford Motor Company to establish or study the possibility of establishing company-sponsored HMOs.

During the evolution of the group practice HMO, another type of HMO emerged in 1954 when the San Joaquin County Medical Society established the San Joaquin Foundation for Medical Care. The Foundation for Medical Care (FMC) is an autonomous corporation sponsored by local, state, or county medical societies; medical society members may voluntarily request membership by contracting to abide by the FMCs operating guidelines. FMCs maintain the fee for service practice of medicine and solo practice while adopting the prepayment mechanism which is essential to the HMO concept. FMCs however are only one form of what is more broadly termed an individual practice association (IPA). What distinguishes the IPA type from other HMOs is that members select a primary care physician from participating physicians who provide services from their own offices instead of a common facility.

ESSEX COUNTY MEDICAL SOCIETY HEALTH ORGANIZATION

Following four years of committee work and soul searching, a group of physicians† incorporated the Essex County Health Organization on January 11, 1977, under the sponsorship of the Essex County Medical Society.

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The HMO Act (P.L. 93-222) became law on December 29, 1973 when the Essex County Medical Society (ECMS) was in the midst of developing an IPA to relate specifically to a Newark project, "COMPRE-HEALTH," then known as the "Medicaid Waiver Project." This never was successful, so the ECMS immediately applied for a Federal grant under 93-222, to develop its own HMO/IPA and our application for the funding of a feasibility study was denied.

Meanwhile, Crossroads Health Plan (CHP) obtained funding for a feasibility study for a closed-panel HMO and decided it was not feasible. They then asked the ECMS to sponsor the development of an IPA under their revised grant. This IPA would have an all-physician board and contract with CHP for physician (M.D. and D.O.) services.

This cooperative interest in establishing a new health delivery system in Essex County eventually led to an educational program to inform individual medical society members; consultants were retained to advise the medical society on various planning and developmental issues presented by Crossroads. An important preliminary step to this cooperative HMO effort between the management and board of Crossroads and the Essex County Medical Society was the mutual agreement that physicians appointed by the ECMS would constitute a majority of the Crossroads 21-member Board of Directors.

Although many IPAs organized in the nation are structured as profit-making corporations, ECHO is organized as a non-profit corporation because this status provides the maximum flexibility to facilitate physician membership. There are no stockholders who control specific shares but every physician enters ECHO on an equal basis and pays the same membership fee to defray operating expenses.

New Jersey statutes would prevent a for-profit IPA from contracting directly with other health care providers such as skilled nursing homes, ambulance services, clinical laboratories, and others. Since ECHO members collectively would be at financial risk for these services, a non-profit organization enables the membership to determine the necessary contractual agreements with the other providers.

ADVANTAGES

The advantages of the ECMS sponsoring an individual practice association were evident because it permitted physicians voluntarily to join an HMO which would:

1. Preserve the fee-for-service approach.
2. Preserve the physician's patterns of practice and referral.
3. Accommodate all types of medical practice from solo to clinic.
4. Afford those enrolled in the HMO free choice of IPA physicians who render care in their own offices and hospitals.

A "negative" incentive was to set up an IPA to offset the loss of up to ten percent of patients who might elect to join a closed panel HMO, one of which was developing in Essex County (Central Essex Health Plan—located near Orange Memorial Hospital). Because of Federal regulations, more and more employees will be electing an HMO benefits package, which means they no longer will have private health insurance. Taking the IPA route interferes least with the traditional physician-patient relationship.

USUAL AND CUSTOMARY

My first job with ECHO was to serve as Chairman of the Physicians Fee and Reimbursement Committee. This committee of twenty physicians decisively refused to accept the fee schedule of other groups and insisted on developing a local "Usual, Reasonable and Customary Fee Schedule" (URC) by sending out a fee survey to all 250 physicians who were IPA members at that time. ECHO employed American Health Systems to evaluate the questionnaire results; these procedures were ratified by the IPA membership at our first annual meeting.

We decided to pay "usual and customary fees up to the 90th percentile, with 15 percent withheld (the "at-risk portion)" pending good contract experience. The ECHO member could expect payment for services billed to the IPA ten days following the close of the books each month, unless his treatment or fee fell outside of our model treatment guidelines of URC limits and was therefore scheduled for peer review. Fees over the 90th percentile are paid at the 90th percentile.

PEER REVIEW

On our request, our local PSRO (EPRO) developed a system of Peer Review and Model Treatment Guidelines for use by our IPA. The ECHO Peer Review and Quality Assurance Committee approved this entire system, which is the heart of the IPA concept. Unless IPA physicians continually scrutinize all claims which fall outside our local established parameters, member physicians would not be reimbursed any of the 15 percent-risk monies which were withheld. Beyond that, the negotiated contract with the HMO could fail through overutilization.

ECHO COMMITTEES

The organizational responsibilities of the IPA are carried on by additional committees such as Medical Practice, Legal/Contracts, Financial/Administrative Appeals, and Membership. The Medical Practice Committee already has faced many knotty problems such as how the IPA should handle and reimburse laboratory services, handle emergency services, and pay covering physicians who were not ECHO members. It is the duty of the Financial Administrative Committee to advise on budgets and arrange for the handling and reimbursement of risk monies and the management of all ECHO funds. The appeals mechanism has been established to handle all grievances from ECHO members and HMO enrolled patients.

PHYSICIAN RESPONSE

The Membership Committee's initial efforts, plus the sponsorship of the Essex County Medical Society, led to an early enrollment of 250 physicians. After six months of operation we have grown in membership so that we now have enrolled one third of the 1850 practicing M.D.s and D.O.s in the Essex area. We are witnessing continued growth as more and more doctors are contacted by HMO patients.

CROSSROADS HEALTH PLAN

Led by Executive Director Robert Detore, Crossroads Health Plan actively has been marketing and enrolling HMO patients through employee groups. We now are delivering comprehensive health care to 1500 patients who enrolled after six months of marketing effort despite tight competition from other health insurance coverages. Crossroads received federal qualification on March 17, 1978 and could not market prior to that time. A three-year projection of 18,000 enrolled has been made. Films and materials for prospective enrollees have been developed and efforts now are directed toward the larger employers in the area.

Both CHP and ECHO are non-profit corporations under New Jersey law. ECHO has an all M.D. and D.O. Board. The CHP Board of Trustees is comprised of half physicians and half consumers and representatives of other groups such as labor and industry.

CROSSROADS PROGRAM

The principal aspects of the Crossroads Program are as follows:

Subscribers—Potential Crossroads subscribers are Essex County residents who are employed by major corporations in the area. The Crossroads marketing staff actively has been presenting our program both to employers and to their employees.

Benefit Package and Premium—The Crossroads benefit package conforms to federal and state regulations and the competitive needs of the marketplace. Subscribers will have a choice between basic option and major option benefit packages. These benefits are under the coordination of the primary care physician, who remains the principal decision maker in determining the appropriateness of care and all referrals.

Contractual Relationships—Crossroads contracts on a yearly basis with the Essex County Health Organization for the provision of physician services, including specialists, to its subscribers. Crossroads also contracts with area hospitals for inpatient and ancillary services. The Essex County Health Organization holds the contracts with other providers of care such as skilled nursing facilities, ambulance services, x-ray and diagnostic laboratories. These contracts also are negotiated on a yearly basis.

Reinsurance—Through reinsurance policies with Mutual of Omaha, Crossroads has financial protection for catastrophic illness, out-of-service area emergency services, inpatient hospital overutilization, and insolvency. This program also permits a member who leaves the Essex County area to maintain uninterrupted coverage by converting to a United Benefit Life Insurance Company Standard Group Conversion Policy (Mutual of Omaha).

Financial—Principal sources of Plan income include premiums earned, income from coordination of benefits and interest earned on available cash resources. The working capital position of the Plan is further strengthened by the availability of proceeds from a $2.5 million federal loan subsidy program. Crossroads has provided a contingency reserve from the loan drawdowns to serve as a safeguard against overutilization of medical services and unit cost variances. Crossroads has demonstrated the viability of its Plan and adequate financial security against the risk of insolvency to federal and state regulatory agency reviewers.

Crossroads Health Plan is now operational after successfully passing the stringent requirements necessary to be designated as a federally qualified and state certified HMO.

Crossroads offers an alternative health care delivery system which is responsive to the interests of both consumers and providers and which includes the following characteristics:

(1) Free choice of participating physician for the patient.

(2) Participating physicians continue to practice in their own private offices and treat both CHP and non-CHP patients.

(3) Participating physicians are paid on a fee-for-service basis. For his part, the participating physician agrees for a one-year period to:

(a) Accept Crossroads members as patients and to provide needed services to these patients on the same basis as non-Crossroads patients.

(b) Refer Crossroads members only to medical specialists and consultants who are also physician participants in the Plan, and to hospitalize Crossroads members only in hospitals which are participating hospitals.

(c) Maintain such records as are necessary to disclose fully the extent of services provided to Crossroads members, and to provide ECHO and Crossroads with the necessary information for the proper administration of the Plan.

(d) Submit claims for services rendered to Crossroads members at least monthly, based on the physician's usual, customary, and reasonable fees pertaining to non-CHP members, but not to exceed the 90th percentile of the URC in Essex County.

(e) Accept payment from the Plan as full payment for services rendered, except certain allowable co-payments and for those services not covered by the member's contract which may be billed directly.

(f) Cooperate fully with any utilization review, peer review, or other programs established by the Plan to promote high standards of medical care, and to control unnecessary and/or inappropriate utilization of services.

(g) Accept a portion of the financial "risk" of providing covered services to CHP members on a prepaid basis. Such risks are limited to 15 percent of the claims submitted.

Past HMO experience has indicated that cost reductions can be realized under these circumstances. Since the participating physicians share in the distribution of these cost reductions, they also share a portion of the financal "risk" involved in providing these services.

Crossroads guarantees to make an initial payment of 85 percent of the claim submitted by the physician (less any co-payment or other disallowances) not later than the 10th working day of the following month. This will eliminate bad debts and improve cash flow. The remaining 15 percent will be placed in an ECHO physician-controlled trust fund as reserve against overutilization. At the end of the year the surplus balance in the risk pool will be returned to the participating physicians. In addition, the participating physicians will share in the distribution of the surplus balances of the Physician Services and Inpatient Services Funds. The Physician Services Fund is the portion of premium assigned to medical services and against which all physicians' charges are applied. The Inpatient Services Fund is the hospital component of premium from which all hospital charges are paid. Thus it is conceivable that the physician will receive more than 100 percent of his usual, reasonable, and customary fees at year end due to savings.

FEDERAL COMMITMENT

HEW Secretary Joseph Califano repeatedly has backed the HMO concept as an alternate health care delivery system stressing quality care at reasonable cost and competitiveness with the traditional modes of medical practice. Representative Peter A. Rodino, Jr., encouraged the development of our program to supply comprehensive health care to Essex residents. The Carter Administration and Congress have shown its commitment to this type of program in its recent allocation of more funds for HMO development.

OUR HMO/IPA FUTURE

Our growth now depends upon successfully marketing a competitive benefit package and the continuing participation and understanding of Essex County physicians. From the

patients' viewpoint, the attractiveness of the sales package is dependent upon a goodly number of local physicians becoming members and remaining ECHO physicians. This offers them a large selection with little disruption of present medical care patterns. This process of meeting physician expectations and addressing their concerns while maintaining an awareness of the health benefit market is vital to our success. In the establishment of this HMO, we have addressed the hot issue of voluntary cost containment in a positive and creative way.

SUMMARY

ECHO and Crossroads exemplify how physicians can assume a position of leadership in the early stages of designing an alternative health delivery system. By assuming a primary role in the embryonic stages of the IPA-HMO, Essex County physicians have played a dynamic part in guaranteeing that the Crossroads-ECHO partnership satisfies the needs of both patients and physicians.

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Left Atrial Myxoma: Echocardiographic Identification*

MICHAEL V. COHEN, M.D., Bronx, New York**

Rheumatic mitral stenosis is the most common cause of obstruction to blood flow across the mitral valve. However, when evaluating a patient for mitral stenosis, the other less common causes always should be entertained, even if only for a moment. Thus, congenital mitral stenosis, or triatriatum, left atrial ball valve thrombus, and left atrial myxoma each can simulate many of the symptoms and clinical signs of rheumatic mitral stenosis. Precise identification of the etiology of the valve obstruction is necessary because of different therapeutic considerations. This report details the case of a patient referred with a presumptive diagnosis of rheumatic mitral stenosis in whom echocardiographic examination established the correct diagnosis of left atrial tumor.

Case Report—A 60-year-old housewife denied any knowledge of heart murmurs or rheumatic fever and claimed to have been able to perform all activities without limitation until one and a half years prior to her admission to Montefiore Hospital and Medical Center (MHMC). She began to experience exertional dyspnea and burning precordial pains inconsistently relieved by sublingual nitroglycerin. Occasionally these symptoms were accompanied by brief episodes of palpitations. She never complained of lightheadedness or syncope and denied any postural influence on her symptoms. There was no history of fever, arthralgias, or weight loss. While sitting her pulse was 100 beats per minute and regular and blood pressure was 140/80 mm Hg. There was little change when the patient was supine. The lungs were clear. The apical cardiac impulse was not displaced, but there was a prominent left parasternal lift. The intensity of the first heart sound was increased, and P_2 was accentuated. A high-pitched opening snap occurring approximately 0.12 sec after A_2 was audible along the left sternal border. A grade 1-2/4 apical diastolic murmur with pre-systolic accentuation was heard. There was no systolic murmur. Cardiac x-ray series revealed left atrial enlargement. The electrocardiogram showed sinus rhythm and no diagnostic abnormalities. The clinical impression was mitral stenosis, and the patient was referred to MHMC for non-invasive studies.

Phonocardiography revealed a loud S_1 with a Q-S_1 interval exceeding 0.08 sec. A prominent extra-diastolic sound, not

*Edited by Sidney R. Arbeit, M.D., deceased, and Ira L. Rubin, M.D., New York. Dr. Arbeit was Associate Clinical Professor of Medicine, New Jersey Medical College, CMDNJ; attending physician and emeritus chief, division of cardiovascular diseases, Jersey City Medical Center; attending physician and chief of cardiology, Jewish Hospital, Jersey City; associate attending physician, division of cardiology, Montefiore Medical Center, Bronx, New York. Dr. Rubin is Clinical Professor of Medicine, Einstein College of Medicine, New York; attending physician, division of cardiology, and chief of electrocardiographic service, Montefiore Medical Center, Bronx, New York.

**Dr. Cohen is a member of the Division of Cardiology, Department of Medicine, Montefiore Hospital and Medical Center and the Albert Einstein College of Medicine, Bronx, New York. He is the recipient of a Research Career Development Award HL 00281.

corresponding with the 0 point of the apexcardiogram, was recorded 0.09-0.10 sec after A_2 along the left sternal border and at the apex. The contour of the apexcardiogram was unremarkable except for a notch during isovolumic contraction. The carotid pulse tracing was normal.

Echocardiography demonstrated evidence of a mass behind the mitral valve. As seen in Figure 1 the normally clear space behind the anterior mitral leaflet was filled with multiple parallel echoes throughout systole and diastole. The abnormal echoes extended posteriorly into the left atrium. The extent of these abnormal echoes is better appreciated in Figure 2. Here the transducer on the patient's chest wall was rotated so that the sound waves were initially directed at the aortic root and left atrium (left) and then at the base of the left ventricle (right). The left atrium behind the aortic root is obviously filled with abnormal echoes which persist and are seen behind the mitral valve and extending into the left ventricle. The diagnosis of left atrial myxoma was made and the patient was admitted to the hospital for cardiac catheterization.

The pulmonary artery pressure was 80/40 (54) mm Hg. The mean pulmonary capillary wedge pressure was 35 mm Hg, and the mean gradient across the mitral valve was 25 mm Hg. The calculated mitral valve area was 0.8 cm^2. Pulmonary angiography and left ventriculography revealed a radiolucent globular structure originating from the left atrial septum which protruded into the left ventricle during diastole. At the time of left thoracotomy a 6x5x3 cm myxomatous mass was removed from the left atrial septum. The mitral valve itself was normal. The patient made an uneventful recovery. Shortly after the surgery repeat echocardiographic examination (Figure 3) demonstrated a normal mitral valve without evidence of persistent tumor. A follow-up study a year and a half later also was normal.

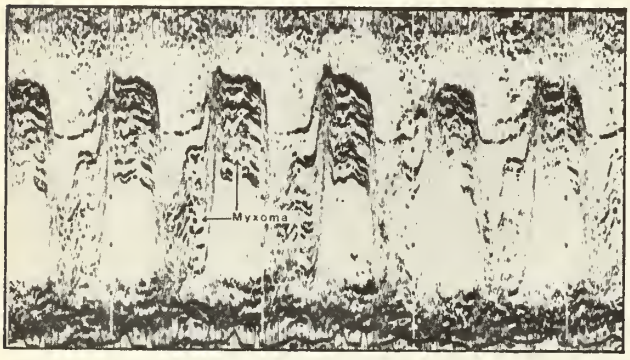


Figure 1 – Echocardiogram demonstrating multiple echoes from a large left atrial myxoma behind the anterior mitral leaflet. The small triangular-shaped zone free of echoes immediately following opening of the anterior mitral leaflet is created because tumor inertia delays descent of the mass into the mitral orifice for approximately 40 msec after the onset of diastole.

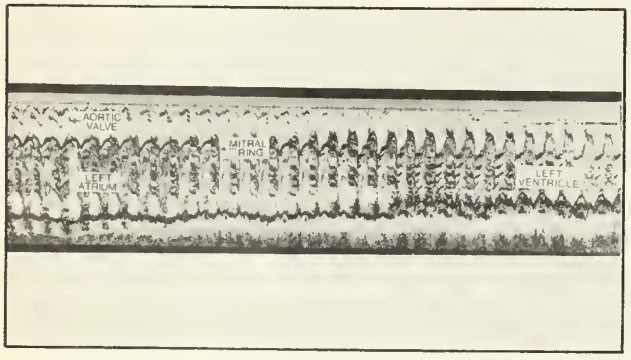


Figure 2 – Cardiac sweep from aortic root to body of left ventricle demonstrating extent of left atrial mass. At the left of the figure, the mass can be seen to be filling almost the entire left atrium. As the transducer is directed more caudally the tumor also can be seen to fill the area behind the mitral valve and protrude into the left ventricle during diastole.

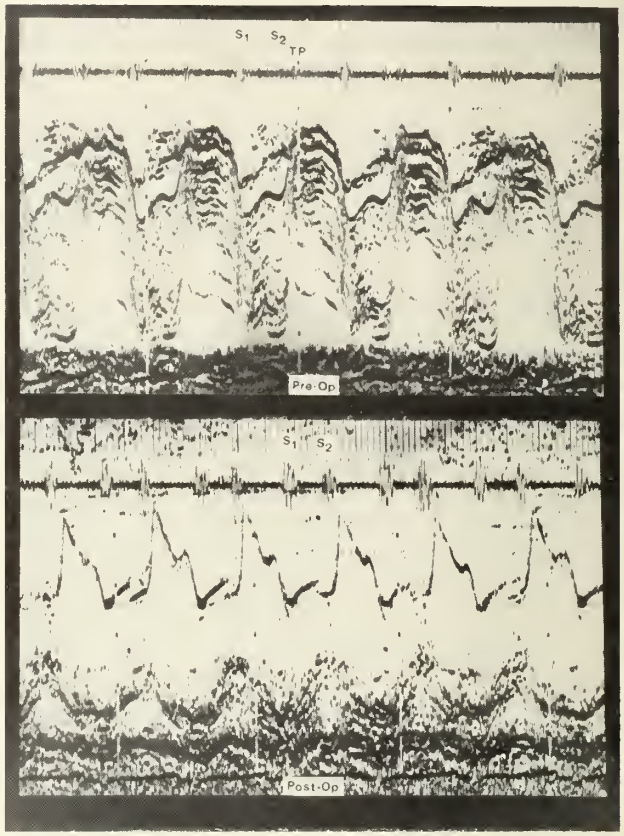


Figure 3 – The pre-operative echocardiographic study demonstrates the left atrial mass behind the mitral valve. A simultaneous phonocardiogram shows the extra diastolic sound (TP = tumor plop) which accompanies movement of the tumor into the mitral orifice at the beginning of diastole. This sound frequently is confused with the opening snap of mitral stenosis. After removal of the tumor the mitral valve echogram is normal and the extra diastolic sound has disappeared.

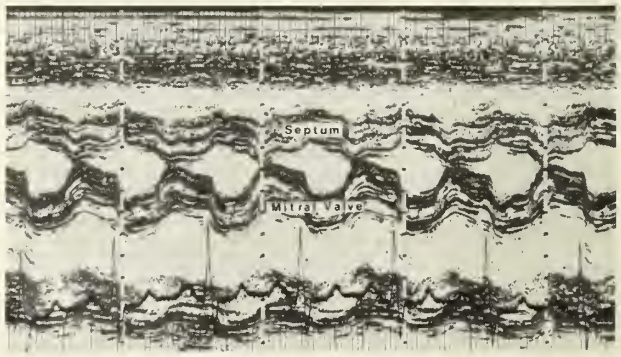


Figure 4 – Echocardiogram from a patient with rheumatic mitral stenosis and a heavily calcified mitral valve. Although there are multiple parallel echoes filling the region behind the anterior leaflet, these extra echoes do not extend into the left atrium and there is no clear zone immediately following anterior leaflet opening, thus effectively excluding the possibility of left atrial tumor.

DISCUSSION

The echocardiographic examination in this patient unequivocally demonstrated a left atrial tumor as the cause of her symptoms and physical signs. The clinical distinction between rheumatic mitral stenosis and left atrial tumor is more than academic. Whereas patients with mitral stenosis may be treated conservatively until symptoms significantly limit their daily activities, even the asymptomatic patient with an atrial tumor must have surgical excision to prevent systemic tumor emboli, intractable heart failure, and even sudden death.

The echocardiographic appearance of left atrial tumors is well demonstrated in Figure 1. Multiple echoes fill the area behind the mitral valve in both systole and diastole. With smaller tumors the extra echoes may be restricted to diastole or even only a part of diastole. The anterior mitral leaflet swings anteriorly at the beginning of diastole. However, because of its inertia, the tumor does not drop into the mitral orifice for approximately 0.04 sec following valve opening thus creating a small triangular-shaped clear space following the initial movement of the anterior leaflet. This clear zone is demonstrated particularly well in Figure 1, and helps to distinguish atrial myxoma from other conditions producing multiple echoes behind the anterior mitral leaflet. The echocardiogram of a patient with mitral stenosis and a heavily calcified mitral valve is presented in Figure 4. Calcium is a particularly good sound wave reflector and deposits in the anterior mitral leaflet account for the multiple echoes. However, no triangular-shaped clear space is evident immediately following opening of the anterior leaflet, thus making the diagnosis of atrial myxoma in this case unlikely. Mitral valve vegetations also may produce multiple echoes behind the anterior mitral leaflet, but again no clear zone will be evident.

Left atrial myxoma unlike other causes of increased mitral valve echoes can be visualized clearly in the left atrium. Whenever an atrial tumor is suspected a continuous cardiac sweep from the aorta or base of the heart towards the apex should be part of the routine examination. As seen in Figure 2 the tumor in this patient practically filled the entire left atrium. Neither vegetations nor leaflet calcium deposits would be expected to produce such atrial echoes. As the transducer is swept caudally, it is apparent that the tumor in this patient extends into the left ventricle during diastole.

The postoperative study presented in Figure 3 demonstrates normal motion of the anterior mitral leaflet. Since the tumor originates from the atrial septum the mitral valve would be expected to be normal. Serial examination of all patients after tumor removal is worthwhile. Although the pathologist may have difficulty classifying a tumor as benign or malignant, recurrence of multiple echoes behind the mitral valve would prove the tumor's malignant character.[1]

All patients with the suspected diagnosis of mitral stenosis should be evaluated routinely with echocardiography. Even a prior history of rheumatic fever should not dissuade the physician from requesting this examination. With this simple non-invasive test the referring diagnosis easily can be confirmed. Perhaps more importantly, those patients with left atrial tumors requiring more urgent treatment can be identified.

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Nutrition and Aging

MYRON WINICK, M.D., New York

Currently, more than twenty million Americans are 65 years of age or older. There are nearly 25,000 nursing homes in the United States containing almost a million and a half beds—more nursing home beds than hospital beds. This population, our "senior citizens," is growing rapidly every day. If aging is a problem, then it is increasing in magnitude; society, of course, by focusing on health issues at younger ages, is trying to lengthen life. Thus, we have within our society, as well as many other so-called advanced societies, what has become a contradiction, which is often difficult to deal with: that is, more old people and, therefore, more problems of the elderly. There are two ways in which one may deal with this problem: 1) reducing the burdens that the elderly must bear and 2) curtailing the process during the early and middle years that leads to the physiologic state that we presently define as "old age." In a sense, we must seek ways of not slowing but actually changing the aging process, as well as seek methods to structure the environment of those reaching old age to provide them with optimal health and well-being. Nutrition is an important component of both of these approaches.

FEEDING THE ELDERLY

A number of physiologic changes occur with aging, which should be understood before an adequate nutritional program can be developed rationally. From a nutritional standpoint, the most important of these changes relates to the renal, neuromuscular, and gastrointestinal systems. Under resting steady-state conditions, most of the components of the "internal environment" are maintained at fairly normal levels even at quite advanced ages; however, renal function clearly deteriorates with age. Table 1 outlines the major renal changes, which occur with aging.

Table 1

a. In humans, the renal blood flow and glomerular filtration rate decline with age.
b. In humans, the T_m for glucose and PAH declines with age.
c. In humans, the ability to form either a concentrated or dilute urine decreases with age.
d. In rats, there is a decline in the number of nephrons with age.

Most of the functions outlined decrease about 0.6 percent per year in the adult.

Gastrointestinal function also declines with aging. (Table 2)

Table 2

a. In humans, the ability of parietal cells to secrete hydrochloric acid declines with age. In general, there is a reduction in the secretory ability of the digestive glands.
b. In humans, xylose absorption is normal until 80 years of age.
c. In humans, calcium absorption decreases beyond 65 years of age.

Therefore, a number of processes basic to the digestion and absorption of nutrients are impaired during aging, whereas others, such as glucose absorption, are unaffected.

The motor function of man also declines with age. There is a generalized weakness probably due both to a decline in the number of functioning muscle fibers and in the contractile process itself. In addition, body composition also changes during aging. Bone loss is a general phenomenon beginning by the fifth decade in both men and women. This condition progresses more than twice as fast in the female as in the male. Although it generally has been believed that there is a loss in lean body mass and an increase in the adipose tissue mass with age, the data have been gathered in cross-sectional studies. Recent data from longitudinal studies (i.e., collected on the same patient over a period of time) suggest that lean body mass does not decline with age but rather that individuals with a smaller lean body mass tend to live longer. Finally, in a series of interesting studies in rats, it has been shown that adipocyte number actually increases very late in life. The significance of these findings remains unknown but the suggestion is that old age is accompanied by relative obesity, due to an actual hyperplasia (increase in cell number) within the adipose depot.

The changes outlined above suggest the need for careful evaluation of nutritional requirements for the elderly, especially in relation to certain specific nutrients. For example, the decrease in muscle mass suggests that protein and amino acid requirements may change. The reduced calcium absorption coupled with bone resorption suggests a reevaluation of the calcium and phosphorus requirements. The decreased secretion of hydrochloric acid coupled with general gastric atrophy suggests an increased iron and perhaps vitamin B_{12} requirement. Unfortunately, careful research aimed at determining requirements of most nutrients specifically for the

*Reprinted with permission of *Contemporary Nutrition* 2:6 (June) 1977, a newsletter from the Nutrition Department of General Mills, Inc., Minneapolis. Dr. Winick is Director, Institution of Human Nutrition, College of Physicians and Surgeons, Columbia University, New York.

elderly is just beginning. At present, neither current United States nor international dietary allowances have included separate recommendations for a single nutrient for the elderly.

Recently, studies have begun seeking to determine the protein and amino acid requirements of the elderly. In humans, the amount of total body protein synthesized daily declines with age. In addition, the relative amounts of protein synthesized by various organs is redistributed. Visceral tissue makes a greater overall contribution to total body protein synthesis in the elderly than in the young adult. From these observations one is tempted to predict that the total protein requirement for the elderly should be less per unit of body weight. Actual measurements of protein requirements using a variety of techniques suggest that the minimum protein needs of healthy adults do not change significantly with advancing age. Thus, on the basis of current knowledge, the recommended intake for healthy males is 56g. and for females it is 46g. This appears to be adequate for most healthy elderly individuals.

Even less is known about amino acid requirements than about total protein requirements. At present, preliminary data would suggest that the requirements for the amino acids threonine and tryptophan are similar to those for young adults when expressed per unit of body weight. Literally nothing is known about the other amino acids.

Medical and health professionals then are left to make decisions about protein and amino acid requirements for the elderly based on limited data. Therefore, our decisions at best only can be "educated guesses." Based on our present state of knowledge, I would recommend that protein intake for the elderly consist of amounts adequate to meet the minimum daily requirement of the healthy young adult but not a great deal above this. In addition, the quality of the protein should be such that it is rich in the essential amino acids and should be supplied in a form that is easily digestible. Thus, fish, soft cheese, lean meats and fowl as well as certain vegetable proteins would be good choices. Since, as previously stated, hydrochloric acid in the stomach may not be secreted properly and the intrinsic factor may be low, the elderly may be more prone to anemia due both to poor iron absorption and poor absorption of vitamin B_{12}. Therefore, foods rich in iron and vitamin B_{12} are indicated. These include red meat, liver, and fortified cereals. Requirements for other vitamins and minerals have not been established for the elderly; therefore, we can only provide what is adequate for the young adult. Calcium, however, may be chronically deficient in older individuals.

At present, there are two schools of thought as to the nature of osteoporosis and peridontal disease, two extremely common and often debilitating diseases of older people. One group of investigators believes that they are two totally different diseases, osteoporosis being a slowly progressing degenerative disease of bone and peridontal disease being a chronic infection of the gums with erosion of the underlying bone, loosening, and finally loss of teeth. A second group of investigators feels that they are both the same disease arising from a chronic, long-term calcium deficiency, which slowly drains both the long bones and the jaw bones and results in a propensity toward long bone fractures and loss of teeth from their bony sockets.

Both groups agree that calcium treatment alone will do little for the patient with osteoporosis. However, those espousing the chronic calcium deficiency etiology believe that calcium supplementation before osteoporosis develops will prevent or at least delay the process. They feel that the recommended dietary allowance is much too low and that adults should take in a minimum of one gram of calcium daily and preferably more than that. In addition, based on experiments with peridontal disease in horses and with limited trials in humans with peridontal disease, these individuals feel that calcium therapy in high doses will reverse the process in the jaw bones and lead to the healing of the peridontal tissues and strengthening of the teeth within their sockets.

My own feeling based on my understanding of the data is that osteoporosis may in part be due to a chronic calcium deficiency because of the low intake of calcium by modern society. I would, therefore, recommend that foods rich in calcium be recommended during adult life as well as childhood and that patients, who are middle-aged and who have symptoms and/or x-ray findings of osteoporosis, be supplemented with a gram a day of calcium in the form of gluconate. While I am not convinced that this will prevent progression of the disease, I feel that the risk of this form of "prophylaxis" is minimal and the potential benefits are high enough to warrant this approach.

With peridontal disease, the data are difficult to interpret. Currently, I feel that this is a disease of multiple etiology and that some forms may respond to calcium treatment. Since it has been reported in some cases to have considerable benefit with objective changes in the x-ray picture, I would recommend short-term treatment (three to six months) to be used. Again, not because I am convinced of its efficacy but because the potential benefits far outweigh the risks.

Certain diseases associated with the gastrointestinal tract are more common in the elderly—especially the elderly of our more affluent societies. Diverticulitis, gall bladder disease, and cancer of the colon are among these diseases. There is evidence which suggests that all three of these may be related to the amount of fiber in the diet.

The data are strongest with diverticulitis. Low fiber intake increases the prevalence of this disease. This is probably because of the decreased transit time it produces and the subsequent hardening of stools and constipation which follows. Since constipation is a serious problem in many older people, I would suggest an increase in the fiber content of their diet. This is probably best accomplished by increasing their bran intake.

SUMMARY

There is much we still must learn about nutrition and aging but there are certain practical things which we can do now to improve the nutrition of the elderly. These include eating a balanced diet, relatively low in fats, high in fiber and, particularly, rich in iron, vitamin B_{12}, and calcium.

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Winick M: *Current Concepts in Nutrition, Vol. 4, Nutrition and Aging.* New York, John Wiley, 1976.

Kritchevsky D, Story JA: Dietary fiber and cancer. In: *Current Concepts in Nutrition, Vol. 6, Nutrition and Cancer.* New York, John Wiley, 1977.

Wynder EL, Reddy BS: Diet and cancer of the colon. In: *Current Concepts in Nutrition, Vol. 6, Nutrition and Cancer.* New York, John Wiley, 1977.

213th Annual Meeting
May 12-15 — Atlantic City

Headquarters Hotels

Holiday Inn—Boardwalk Regency

(See **housing application—p. 232, this issue**)

DAILY SCHEDULE

Friday, May 11, 1979
3:30 p.m. —Board of Trustees

Saturday, May 12, 1979
8:00 a.m. —Registration Opens
9:00 a.m. —Message Center Opens
11:30 a.m. —Meeting—NJ Committee on Trauma
11:30 a.m. —Luncheon—NJ Orthopaedic Society
12 noon —Scientific and Informational Exhibits Open
12 noon —Luncheon—NJ Committee on Trauma
1:00 p.m. —Annual Spencer T. Snedecor Trauma Oration
2:00 p.m. —Motion Picture Theatre
2:00 p.m. —House of Delegates
3:30 p.m. —Reference Committees: Constitution and Bylaws, "A", "B", "D", "G"
6:00 p.m. —Reception for Alumni—Jefferson Medical College

Sunday, May 13, 1979
7:15 a.m. —Annual Prayer Breakfast
8:00 a.m. —Registration Opens
8:00 a.m. —Scientific Session on Oncology
9:00 a.m. —Message Center Opens
9:00 a.m. —Scientific and Informational Exhibits Open
9:00 a.m. —Scientific Sessions:
Allergy, Emergency Medicine, Medicine
Dermatology, Obstetrics and Gynecology
Family Practice, Pediatrics
Neurosurgery and Neurology
Psychiatry
9:30 a.m. —Meeting—NJ Academy of Ophthalmology and Otolaryngology
10:00 a.m. —Motion Picture Theatre
10:00 a.m. —Reference Committees: "C", "E", "F", "H"
11:30 a.m. —Luncheon—NJ Academy of Ophthalmology and Otolaryngology
12 noon—Luncheon—NJ State Urologic Society
12:15 p.m. —Luncheon—NJ Chapter, American College of Emergency Medicine
1:00 p.m. —Luncheon—Oncology Society of NJ
1:00 p.m. —Scientific Sessions:
Anesthesiology
Cardiovascular Diseases, Medicine, Nuclear Medicine
Ophthalmology
Otolaryngology
Plastic and Reconstructive Surgery
Urology
2:00 p.m. —Motion Picture Theatre
3:00 p.m. —House of Delegates
4:30 p.m. —Meeting—Widows and Orphans Society
6:30 p.m. —Inaugural Reception Honoring President-Elect and Mrs. Alessi
Hosts—MSNJ and Bergen County Medical Society
All members, official guests, and their wives, and Auxiliary members are cordially invited
—Tickets $5.00 per person
8:00 p.m. —Inaugural Dinner
Hosts—MSNJ and Bergen County Medical Society
Music and Entertainment—Danny Davis and His Nashville Brass
Tickets $30.00 per person

Monday, May 14, 1979
7:00 a.m. —JEMPAC Breakfast
8:00 a.m. —Scientific Session on Orthopaedic Surgery, Family Practice
8:30 a.m. —Registration Opens
8:30 a.m. —Scientific Session on Surgery, Clinical Pathology
9:00 a.m. —Scientific Sessions:
Medicine, Rheumatism
Radiology
9:00 a.m. —Message Center Opens
9:00 a.m. —Scientific and Informational Exhibits Open
9:00 a.m. —JEMPAC Political Action Seminar
9:30 a.m. —Scientific Session on Chest Diseases
10:00 a.m. —Motion Picture Theatre
11:30 a.m. —Luncheon—NJ Society of Physical Medicine and Rehabilitation
12:00 noon —Luncheons:
NJ Chapter, American College of Chest Physicians
NJ Orthopaedic Society
1:00 p.m. —Scientific Sessions:
Gastroenterology and Proctology
Physical Medicine and Rehabilitation
1:00 p.m. —Meeting—NJ Foundation for Health Care
2:00 p.m. —Motion Picture Theatre
3:00 p.m. —House of Delegates
3:00 p.m. —Scientific and Informational Exhibits Close
6:00 p.m. —JEMPAC Wine and Cheese Reception

Tuesday, May 15, 1979
6:45 a.m. —Breakfast Meeting of the "Coalition of Southern NJ County Medical Societies"
Reservations: Mrs. Ruth E. Bunning, Exec. Sec., Burlington County Medical Society, P.O. Box 56, Medford, NJ 08055
7:15 a.m. —Breakfast Meeting—Bergen and Passaic County Medical Societies
Reservations: Mr. George Willis, Exec. Dir., Bergen County Medical Society, 170 Main St., Hackensack, NJ 07601
7:15 a.m. —Breakfast Meeting—Essex County Medical Society
Reservations: Mr. Arthur R. Ellenberger, Exec. Sec., Essex County Medical Society, 144 So. Harrison St., E. Orange, NJ 07018
8:30 a.m. —Registration Opens
9:00 a.m. —House of Delegates
12 noon —Registration Closes
2:00 p.m. —Annual Meeting—Board of Governors of the Medical Inter-Insurance Exchange of NJ
3:00 p.m. —Board of Trustees

DOCTORS' NOTEBOOK

Trustees' Minutes January 21, 1979

A regular meeting of the Board of Trustees was held on Sunday, January 21, 1979, at the Somerset Marriott Hotel, Somerset, New Jersey. Detailed minutes are on file with the secretary of your county medical society. A summary of significant actions follows:

Commission on Professional Health Services . . . Received a written report from President Krueger on his presentation to the Commission on Professional Health Services on issues dealing with the lack of alien, foreign, medical graduates resulting from passage of restrictive legislation (P.L. 94-484, which mandates that such graduates must pass an examination equivalent to Parts I and II of the National Boards and a rigorous English examination).

Cornerstone Club . . . Noted that contributions to the Cornerstone Club total $32,000 and approved a recommendation that one more general mailing be made to the membership encouraging donations.

New Jersey State Medical Underwriters . . . Noted that the State Insurance Commissioner has approved the rate filing made by the New Jersey State Medical Underwriters, Inc. Rates were effective January 1, 1979; for new business the effective date is February 1, 1979.

Mortgage on New Headquarters . . . Approved a recommendation to adopt the following resolution:

Mortgage Financing on New Headquarters

RESOLVED, that the Medical Society of New Jersey mortgage its corporate headquarters and real estate located at 2 Princess Road, Lawrenceville, New Jersey to the Prudential Insurance Company in consideration of said company loaning to the Medical Society of New Jersey the amount of $1.6 million dollars at 10 percent interest, to be repaid during a 25-year period; and be it further

RESOLVED, that the proceeds of this loan be applied to offset the purchase, construction and furnishing of the property located at 2 Princess Road; and be it further

RESOLVED, that the President, the Secretary, and Legal Counsel be authorized to execute and perform any and all acts in furtherance of the above resolution.

Basic Science Structure in South Jersey . . . Reaffirmed MSNJ's original position on the basic science facility as set forth in the following statement prepared by the Council on Medical Services and approved by the Board on September 17, 1978:

1. That there is need to increase the basic science capability by 100 students so as to graduate 400 new physicians in New Jersey each year.
2. That the existing basic medical science facilities in Piscataway and Newark should be expanded.
3. That for the present there should be no establishment of a basic science facility in South Jersey; if it is necessary ultimately to establish such a facility, it should accommodate both osteopathic and allopathic students.

Note: The above action was precipitated by the suggestion of the Coalition of Southern New Jersey Medical Societies and the Camden County Medical Society that MSNJ did not support the Coalition's position of opposition to the establishment of a basic science structure in southern New Jersey. Notice of the Board's position (9/17/78) (which coincided with that of the Camden County Medical Society) was sent to the Board of Trustees and to the President of the College of Medicine and Dentistry of New Jersey, to Governor Byrne, to the Chancellor of Higher Education, and to the President of the Camden County Medical Society. In addition, a communication went to MSNJ's lobbyist requesting that the legal counsel for the Governor and the leadership of the Senate and Assembly be apprised of MSNJ's position on the basic science facility in southern New Jersey. This position was reiterated by President Krueger in his presentation to the New Jersey Commission on Professional Health Services on January 18, 1979. Dr. Krueger appeared also before a Board of Higher Education subcommittee, which is studying the "future scope and cost of health professions' education in New Jersey," to present MSNJ's views on the subject. It was the consensus of the Board that the position of the Society on the South Jersey facility had been articulated clearly to the appropriate officers of CMDNJ and to the state government.

Podiatry Services . . . Tabled consideration of the Hospital Association's position on podiatry services within the hospital pending input from the American College of Surgeons, the New Jersey Orthopaedic Society, and other interested groups.

Diagnostic Related Group (DRG) Reimbursement Experiment . . . Directed that an inquiry be addressed to the Commissioner of the Department of Health as to the status of the pilot DRG program and agreed to support the Hospital Association in its request for a hearing, if the program has passed beyond the pilot stage and is, in fact, now being implemented.

Preadmission Testing Office Consultation Program (PATOC) . . . Adopted the following amended version of a recommendation proposed by the Council on Medical Services which suggested expanding the program to include the entire state:

That the Preadmission Testing Office Consultation Program (PATOC) now being conducted solely in Union County be continued for one year.

Note: The Board endorsed the concept of expansion but agreed that the program should be pursued further on an experimental basis.

Henry J. Austin Health Center . . . Approved the following recommendation from the Council on Medical Services:

That the Medical Society of New Jersey disapprove the application of the Henry J. Austin Health Center (Trenton Neighborhood Health Center) for National Health Service Corps personnel.

Cooper Medical Center Ambulatory Care . . . Approved the following recommendation from the Council on Medical Services:

That the Medical Society of New Jersey approve the application of the Cooper Medical Center Ambulatory Care, Inc. for National Health Service Corps personnel.

Emergency Medical Services Project . . . Directed that when the proposed rules (Department of Health) which would establish a process (in accordance with the Emergency Medical Services Systems Act of 1973) for the designation of emergency medical services regions and hospitals to implement a statewide emergency medical services system, as published in the December 1978 *New Jersey Register* and subsequently revised, are received they are to be referred to the Executive Committee for review and development of a position paper for presentation at the February 1st public hearing held by the Health Care Administration Board.

Health Care Administration Board . . . Received as informative a report on the January 4th meeting of the Health Care Administration Board and directed that a congratulatory letter be sent to John B. Reiss, Ph.D., Assistant Commissioner, Health Planning and Resources Development, on the occasion of his assuming a new post in the Health Care Financing Administration (HCFA) of DHEW.

The items covered in the report included:

1. *Rules on Hospital Reporting Regarding Patient Case Mix*—for use primarily in the DRG project and the prospective rate setting.
2. *Rule on Economic Factor*—the number (percentage) by which rates can be increased for inflation and the methodology by which cost components are considered.
3. *Rules on Designation of Emergency Medical Services Regions and Hospitals* (see Emergency Medical Services item above).
4. *Proposed Amendments to Manual of Standards for Hospitals*—concerning availability of records, verbal orders accepted by physical therapists, and authentication/countersigning of physicians' orders.
5. *Proposed Amendments to Manual for Licensure of Ambulatory Care Facilities—Section 6.3, Prenatal, Postpartum, and Gynecological Services*—these apply to free-standing facilities not affiliated with hospitals.
6. *Unresolved Certificates of Need* for three nursing homes
7. *Existing Health Care Programs*—request for report from the State Department of Health on all existing health care programs and items relating to alternatives for care.

IPA Consortium Project . . . Received a progress report from the NJFHCE on the IPA Consortium Project and noted that the Society's original charge to the Foundation requested a feasibility study to determine the practicality and ability of developing a single statewide IPA/HMO in New Jersey.

National Health Insurance . . . Approved the following recommendation from the Ad Hoc Committee on National Health Insurance which will be presented to the 1979 House of Delegates as a resolution from the Board of Trustees:

That the AMA should proceed with the drafting and introduction of legislation that will embody the following principles:

(a) Require minimum standards of adequate benefits in all health insurance policies sold in the United States with appropriate deductible and coinsurance. (Free choice of physician/patient, and inclusion of as many of the 17 principles of the former AMA policy is indicated.)

(b) A simple system of uniform benefits provided by the federal government for those individuals who are unfortunate enough (through no fault of their own, i.e., age, disability, hardship, and so on) not to be able to provide for their own medical care. (We depart from AMA policy in this section in that we do not call for state and local involvement. In our experience, such a structure produces disparate benefits, and unequal and inefficient administration.)

(c) A nationwide program by the private insurance industry (with government reinsurance if necessary) to make available catastrophic insurance coverage for those illnesses and individuals where the economic impact of a catastrophic illness could be tragic. All catastrophic coverage should have an appropriate deductible and coinsurance to make it economically feasible and to avoid abuse.

(d) A program developed pursuant to these principles should be administered at the state level (utilizing fiscal intermediaries) with national standardization through federal guidelines.

The Ad Hoc Committee also considered Resolution #10—Opposition to National Health Insurance (1978 House of Delegates) which had been referred to the Board and which is being implemented by the foregoing action. To further enhance the Society's position the following recommendation was approved and it was noted that the same materials could be used in conjunction with the public relations efforts on Resolution #13—Governmental Regulation—Health Care Cost Escalation (1978 House of Delegates):

That the Board of Trustees refer the booklet, "Patient Education Packet" to the Council on Public Relations, with the charge that the Council develop an effort to utilize the material either for TV spot announcements or paid air time. Once the concept is developed, requests should be made to have the Private Medical Care Foundation and the AMA underwrite the cost of distribution.

Excessive Fees for Professional Services . . . Referred to the Executive Committee for appropriate response the proposed State Board of Medical Examiners' Rule on Excessive Fees for Professional Services—Proposed NJAC 13:35-6.12

Notice of Generic Drug Availability . . . Referred to the Executive Committee for appropriate response the proposed State Board of Medical Examiners' Rule on Establishing Requirements for Issuing a Prescription Establishing a Requirement of Notice to the Public of Generic Drug Availability—Proposed Amendment to NJAC 13:35-6.6.

Scope of Practice of Podiatrists . . . Noted that the questions posed by Blue Shield relative to the scope of practice of a podiatrist are covered under NJSA 45:5-7.

CME Delinquent Members . . . Noted that as of January 19, 1979, 238 members have not complied with CME requirements; it was further noted that the 1978 House had voted to drop from Society membership those who were not in compliance with 1972 CME requirements as of January 1, 1979. A motion to prepare a news release indicating that delinquent members were being dropped was defeated.

Society of Anesthesiologists . . . Referred a request from the New Jersey State Society of Anesthesiologists for MSNJ's support in the attempt to rectify, through negotiations, the injustices in the area of economics experienced by anesthesiologists through the years to

the Council on Medical Services for study and report to the Board within 60 days. The Chairman of NJSSA's Negotiating Committee was requested to supply the Council with whatever material he had that would assist in the deliberations.

CMDNJ Notes*

Stanley S. Bergen, Jr., M.D.
President

Established at CMDNJ-New Jersey Medical School in 1972, the Department of Neurosciences was designed to integrate basic and clinical neurosciences over the four-year medical school curriculum, as well as at the graduate and postgraduate levels. In the past seven years the department has grown considerably and at present has 13 full-time, and 24 part-time faculty members. The following represent the major academic programs in the department.

Educational: In the first year of medical school, a major course in neurosciences covers in-depth basic neuroanatomy and neurophysiology. Students learn the gross anatomical structure of the human brain, cellular physiology of neurons, interconnections of the various neurological subsystems, and strategies for localizing neurological disease process. Clinical presentations are related to basic concepts as much as possible. In the second year emphasis is on disease processes and their effect on brain structure and function in neuropathology. In the Introduction to Clinical Medicine Course the student is taught how to elicit a neurological history, perform a neurological examination, and interpret the significance of abnormal findings.

The final sequence in undergraduate neurological education comes in the fourth-year, neurology clerkship, which provides students with the opportunity to gain a base of clinical knowledge and skills in neurology. The student learns to improve interview and examination techniques and to determine the etiology and precise localization(s) of the disorder. Emphasis is placed on learning to evaluate and treat neurologic emergencies such as status epilepticus, cerebral herniation and increased intracranial pressure, acute spinal cord compression, acute bacterial meningitis, and coma. In addition, the student becomes familiar with common problems in clinical practice, i.e., headaches, seizures.

The department sponsors the only neurology residency program in New Jersey. This is a fully approved, four-year program based primarily at the new CMDNJ-College Hospital in Newark and the Veterans Administration Medical Center in East Orange. At present there are 22 residents in this program. In addition, the department anticipates approval of a basic science graduate program in the CMDNJ-Graduate School of Biomedical Sciences in the near future. This will enable candidates for the Ph.D. degree to carry out their studies in the department and will add a new dimension to our educational program.

Lastly, the department offers a regular series of postgraduate continuing education workshops, lectures, seminars, and symposia for both the neurologist and non-neurologist. On September 15-16, 1979, an international symposium on multiple sclerosis will be held on our Newark campus.

Research: The department has active, funded research programs in neurovirology, neuroimmunology, behavioral science, and neurochemistry, as well as several other areas. Last year the department published 106 scientific articles.

A major research effort is concerned with the human demyelinating disorders, multiple sclerosis and acute idiopathic polyneuropathy (Guillain-Barre syndrome). Ultrastructural, viral, epidemiologic, and immunologic studies are being carried out by Drs. John W. Prineas, Peter C. Dowling, and myself in an effort to determine the etiology and pathogenesis of these debilitating diseases. Ultrastructural studies have characterized the predictable sequence of morphological changes which are seen in these disorders. Other studies have revealed that cytomegalovirus may be a common infectious cause of the Guillain-Barre syndrome and that serum antibodies to nerve tissue may play an important role in producing demyelination.

Last year, we reported an unusual family in which three sisters living together developed symptoms of multiple sclerosis (MS) in the same year after exposure to a household dog with a transient neurological disorder. This remarkable case history led us to explore the possible relationship between animal exposure and subsequent MS on the premise that an infectious agent might be transmitted as a zoonosis. The retrospective epidemiologic studies in New Jersey showed significantly more indoor dog ownership in the MS population as compared to closely matched controls, particularly in the five-year period prior to onset of MS symptoms. Further, more dogs owned by MS patients developed a neurological illness in this five-year period than dogs owned by the control group. Additional epidemiologic findings in both the Faeroe Islands and Iceland showed a close temporal association between an outbreak of illness in dogs diagnosed as canine distemper and a subsequent "epidemic" of MS in humans. We also found elevated titers of serum canine distemper virus neutralizing antibody in a group of patients with MS as compared to controls. On the basis of these findings, we have proposed that MS may involve transmissions of an infectious agent from animal to man, and that canine distemper or a closely related virus is a likely candidate.

Dr. Barry Levin has focused upon the metabolism of catecholamines, chemical messengers in the nervous system which relay nervous impulses from one nerve cell body to another. A major area of interest is in the transport of catecholamines and their synthetic enzymes within nerve cells of the brain, and how changes in transport may relate to changes in the metabolism and function of these cells. Another major area of interest is in the excretion and metabolism of catecholamines in the plasma of humans and other mammals, as indicators of the function of the adrenal glands and autonomic (vegetative) nervous system. Dr. Levin and his colleagues have found a regular, rhythmical excretion of one such catecholamine, norepinephrine, in monkey and man, and at present are searching for the origin of these rhythms.

Diseases and lesions of the nervous system which affect autonomic function are known to alter plasma levels and metabolism of catecholamines. Some of these which are presently under investigation are spinal cord injuries, disorders of central (Shy Drager syndrome) and peripheral (idiopathic orthostatic hypotension) autonomic pathways, and various other diseases thought to involve autonomic pathways. Their goals are to understand catecholamine metabolism

*This month's column was prepared by Stuart D. Cook, M.D., Professor and Chairman of the Department of Neurosciences at the CMDNJ-New Jersey Medical School.

under normal conditions so as to be able better to evaluate and treat abnormalities of metabolism in disease states in man.

Drs. Allan Siegel, Henry Edinger, and Raymond Troiano have been studying the role of the limbic system in the neurobiology of aggressive behavior. For many years it has been suggested that the limbic system plays a strategic role in the control of aggressive disorders. Dr. Siegel and his colleagues have attempted to identify the behavioral, anatomical, and physiological mechanisms which form the basis for the regulation of aggressive reactions in animals. Their studies have revealed that much of the limbic system normally serves powerfully to suppress aggressive reactions. Regions such as the prefrontal cortex, amygdala, and septal area are examples of such limbic structures.

Other studies have attempted to identify the neuroanatomical pathways associated with limbic inhibition of aggression as well as with the neuronal groups innervated by these inhibitory pathways. In brief, they have shown that all these inhibitory pathways have either direct or indirect connections with the lateral hypothalamus—the primary site from which the types of aggressive behavior studied are elicited. Additional studies currently are attempting to identify some of the basic neurophysiological relationships between the limbic system and hypothalamus in order to further understanding of the neural mechanisms which control aggressive behavior, as well as to provide additional insights concerning how aggressive reactions may become manifest and ultimately controlled in man.

Research in the laboratory of Dr. Benjamin Natelson is directed toward producing and analyzing animal models of psychosomatic disease. The most interesting study to date is that for digitalis toxicity. Guinea pigs are given a subtoxic dose of digitalis. They are then put into an environment in which they previously had been subjected to nociceptive stimuli. Merely being in the environment frequently changes the pattern of their ECG to a digitalis-toxic one. This finding indicates that psychological factors may explain in part the enormous individual variability to constant doses of digitalis.

Special Patient Care Activities: A comprehensive neurological-neurosurgical tertiary care facility has been established at the CMDNJ-College Hospital. This facility contains a neurological-neurosurgical intensive care unit, as well as a fully equipped neuroradiologic suite with a CAT scanner and a modern electrodiagnostic laboratory for electroencephalography, electromyography, and nerve conduction studies. In addition, the electrodiagnostic laboratory performs visual, auditory, and somatosensory-evoked potential testing for evaluating patients with visual, auditory, and spinal cord dysfunction.

As well as offering general neurological services, special diagnostic and therapeutic programs for adults and children with muscular dystrophy, Guillain-Barre, and multiple sclerosis have been established at the CMDNJ-College Hospital under the direction of the department. A similar range of neurological services for veterans also is offered at the Veterans Administration Medical Center in East Orange. This hospital has been designated as an acute inflammatory neurological disease referral center by the Veterans Administration.

Report from the Foundation

Daniel J. O'Regan, M.D.
Medical Director

Enrollment in prepaid health plans in New Jersey nearly has doubled in the past year. In December, 1977, there were about 54,500 subscribers; on January 1 of this year there were nearly 100,000. There are indications that the Rutgers Community Health Plan in New Brunswick may be the fastest-growing HMO in the nation. The Crossroads Health Plan in Essex County may be setting a similar pace for the Individual Practice Association. President Carter's proposed budget for fiscal year 1980 includes: "A major increase in spending to expand prepaid health plans, called Health Maintenance Organizations." To some, this trend is another threat to the traditional practice of medicine. Others emphasize the need for "alternative" delivery systems to help to control costs and to improve accessibility to care. Economists say that there will need to be more "market forces" at work in the health care field. The more alternatives available to the consumer, the more "market" effects will come into play, and the less regulation will be required. Such ideas seem more appropriate to selling shoes than providing medical care, but the thrust cannot be ignored. Planners in government and industry lean toward the closed-panel HMO as being more cost efficient. Given the present atmosphere and the growing interest in our State, NJFHCE has been pursuing the Individual Practice Association (IPA) concept, which more closely approximates traditional fee-for-service, office-based practice. Under the guidance of Ms. Barbara Kempczinska, our IPA/HMO Consortium and Study Group have made great strides in the past year. The essence of physician involvement in prepaid care is, and should always be, professional peer review to monitor the quality and efficiency of the services rendered to the patients. Without the participation of physicians willing to see that the precepts of good medical care are followed properly and in the appropriate setting, and who agree to back up their commitment by the assumption of some risk, prepaid health care will be solely a cost-reducing program. This is why NJFHCE believes that physician input is essential.

213th Annual Meeting

May 12-15, 1979 **Atlantic City**

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Office of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ALLERGY/IMMUNOLOGY—Alan R. Green, M.D., 1315 Ethel Street, Glendale, CA 91207. New York Medical 1971. Partner, associate in single-specialty group, or purchase of practice. Available August 1979.

ANESTHESIOLOGY—Jitendra G. Patel, M.D., 133-52 Avery Avenue, Flushing, New York 11355. M.P. Shah (India) 1973. Board eligible. Any type practice. Available July 1979.

Rolycito A. Chan, M.D., 2121 S. Wolf Road, Hillside, IL 60162. Cebu (Philippines) 1974. Board eligible. Group, partnership, solo. Available July 1979.

CARDIOLOGY—Muljibhai J. Thakkar, M.D., 255-25 75th Avenue, Glen Oaks, New York 11004. Baroda (India) 1972. Solo. Available July 1979.

Masoud Javed, M.D., Deborah Heart and Lung Center, Browns Mills 08015. Liaquat Medical College (Pakistan). Subspeciality, internal medicine. Any type practice. Available July 1979.

FAMILY PRACTICE—Charles A. Joseph, M.D., 99 Morris Avenue, Summit 07901. CMDNJ 1976. Board eligible. Group or partnership. Available July 1979.

Rosario A. Bascon, M.D., 15 St. Andrews Place, Apt. 2-A, Yonkers, NY 10705. Cebu (Philippines) 1967. Board certified. Solo, partnership, group. Available.

INTERNAL MEDICINE—Stephen A. Atlas, M.D., 159 Hawthorne Avenue, Central Islip, NY 11722. Wisconsin. Board eligible. Group or partnership. Available July 1979.

Yugal K. Maheshwari, M.D., 10555 Fondren, Apt. #727, Houston, Texas. J. N. Medical College (India) 1970. Subspecialty, endocrinology. Board eligible. Partnership, associate, single or multi-specialty group. Available July 1979.

Michael D. Burday, M.D., 139-1 Kirkbride Road, Voorhees 08043. Georgetown 1976. Group or partnership. Available July 1979.

Ravi K. Malpani, M.D., 1165 Route 22, Apt. 22, North Plainfield 07061. Osmania (India) 1972. Subspecialty, pulmonary medicine. Board eligible. Group, solo, partnership, or hospital-based. Available July 1979.

John Kassabian, M.D., 25 Burnett Terrace, West Orange 07052. CMDNJ 1974. Subspecialty, pulmonary medicine. Board certified. Any type practice. Available July 1979.

Jerome W. Takiff, M.D., 2917 E. Turquoise, Phoenix, Arizona 85028. Cornell 1973. Board certified. Group. Available.

Kenneth Lipkowitz, M.D., 76 E. Park St., Apt. 26, East Orange, N.J. 07017. Guadalajara 1974. Board eligible. Partnership or group. Available July 1979.

N. M. Koshy, M.D., 8 Slate Creek Dr., Apt. 9, Cheektowaga, New York 14227. Kottayam-Kerala (India) 1971. Board certified. Subspecialty, cardiology. Group, partnership, solo, institution. Availabe July 1979.

Kiril Desai, 108-18 63rd Street, Forest Hills, New York 11375. Baroda (India). Subspecialty, cardiology. Group, partnership, salaried position. Available.

Vennaralagappan Mahadev, M.D., 429 Case Street, Kingsford, Michigan 49801. Madurai (India) 1967. Board certified. Subspecialty, cardiology. Group or solo. Available August 1979.

Harvey M. Cohen, M.D., 8 Deerwood West, Apt. 44C, Irvine, CA 92714. Syracuse 1972. Board certified. Subspecialty, gastroenterology. Solo, group, partnership. Available.

Taha Hamoui, M.D., 9831 Timerwood Circle, Louisville, KY 40223. Damascus (Syria). Board certified. Subspecialty, gastroenterology. Group, partnership, solo. Available July 1979.

Alan J. Sten, M.D., 105 West 13th St., Apt. 15-C, New York, NY 10011. New York Medical (1972). Subspecialty, infectious diseases. Board certified. Partnership or group. Available July 1979.

Pervaiz Rahman, M.D., 240 Yonkers Avenue, Apt. 1-C, Yonkers, NY 10701. Dow (Pakistan) 1972. Subspecialty, gastroenterology. Board eligible. Group, partnership, solo, or institutional. Available July 1979.

Aziz H. Junagadhwalla, M.D., 9 Nixon Court, Apt. 3-J, Brooklyn, NY 11223. Grant Medical College. Subspecialty, pulmonary medicine. Group or partnership. Available July 1979.

NUCLEAR MEDICINE—R. Murty Krishnamsetty, M.D., 101 West 15th Street, Apt. 4-E North, New York, NY 10011. Banaras H. (India) 1971. Special interest, therapeutic radiology. Board eligible. Board certified in T.R. Group or partnership. Available July 1979.

OBSTETRICS/GYNECOLOGY—Khatoon T. Ginwala, M.D., 1817 Rolling Lane, Cherry Hill 08003. Hahnemann. Board eligible. Available July 1979.

Nilima Parekhji, M.D., 12000 Fairhill Road, Apt. 814, Cleveland, Ohio 44120. T.N. Medical (India) 1972. Board eligible. Group, partnership, solo. Available July 1979.

Jeffrey M. Reinkraut, M.D., 40-01 Little Neck Pkwy., Little Neck, NY 11363. Rutgers (CMDNJ) 1975. Board eligible. Group or partnership. Available July 1979.

PATHOLOGY—Mansoora R. Chaudry, M.D., 41-04 Goldblatt Terrace, Fair Lawn 07410. Fatima Jinnah Medical College (Lahore, India). Board certified. Any type practice. Available.

PEDIATRICS—Theordore Sher, M.D., 170 West End Avenue, New York, NY 10023. SUNY—Downstate 1974. Special interest, pediatric allergy. Board eligible. Group or partnership. Available July 1979.

Warren P. Silberstein, M.D., 17 Bartlett Drive, Middletown, NY 10940. SUNY—Downstate. Board eligible. Group practice. Available July 1979.

Vijaya Lakshmi Malpani, M.D., 1165 Route 22, Apt. 22, North Plainfield 07061. Osmania (India) 1972. Board eligible. Group, solo, hospital-based, partnership. Available July 1979.

Bart Asner, M.D., 3949 Los Feliz Blvd., Los Angeles, CA 90027. Pennsylvania 1976. Group or partnership. Available July 1979.

Cecil F. Desai, M.D., 2112 Starling Ave., #5K, Bronx, NY 10462. B.J. Medical College (India) 1968. Board eligible. Salaried position. Available July 1979.

M.S. Krishna, M.D., 32127 Concord Dr., Bldg. 6, Apt. F, Madison Heights, Michigan 48071. Bangalore (India) 1971. Board eligible. Group or partnership. Available July 1979.

PSYCHIATRY—Elaine Nicola, M.D., 117 Grove Street, Ramsey 07446. Northwestern 1970. Board certified. Group, partnership, or clinic. Available.

Herman Berliss, M.D., 110 Boteler Street, Apt. 703, Ottawa, Ontario K1N 8W8, Canada. Case Western Reserve 1974. Board eligible. Adult outpatient. Available July 1979.

PULMONARY DISEASES—Shreedhar B. Shetty, M.D., 116B Davey Street, Bloomfield 07003. Kasturbha (India) 1968. Board eligible. Group. Available July 1979.

RADIOLOGY—Pongthep Thepsongwajja, M.D., 2309 NW 38th Street, Apt. 75, Lawton, Oklahoma 73505. Siriraj (Thailand) 1972. Board certified. Group, partnership, solo. Available.

R. Murty Krishnamsetty, M.D., 101 West 15th Street, Apt. 4-E North, New York, NY 10011. Banaras H. (India) 1971. Subspecialty, nuclear medicine. Board certified (therapeutic radiology). Board eligible (N.M.) Group, partnership. Available July 1979.

SURGERY, GENERAL—William A. Schuckardt, Jr., M.D., 13500 Shaker Road, Cleveland, Ohio 44120. Case Western Reserve 1974. Board eligible. Group, solo, or partnership. Available July 1979.

John L. DeBoer, M.D., 1242 B Cedar Street, Fort Dix, Vermont 1971. Board certified. Single-specialty group, partnership, multi-specialty group, solo. Available June 1979.

David W. Fricke, M.D., 1020 Orr's Bridge Road, Mechanicsburg, PA 17055. University of Pennsylvania 1960. Board certified. Any type, partnership or group practice preferred. Available.

M. Y. Dhar, M.D., 318 East 15th Street, Apt. 8-M, New York, NY 10003. G.R. Medical College (India). Board eligible. Any type practice. Available July 1979.

Stuart A. Engel, M.D., 545 39th Avenue, San Francisco, CA 94121. Johns Hopkins 1974. Group or partnership. Available August 1979.

Michael F. Lane, M.D., 7901 Henry Avenue, Apt. C-411, Philadelphia, PA 19128. SUNY—Downstate 1973. Board eligible.

Group or partnership. Available July 1979.

Donald F. Temple, M.D., 44 Morningside Dr., Apt. 5, New York 10025. Cornell 1974. Board eligible. Group, partnership, hospital, solo. Available July 1979.

SURGERY, ORTHOPEDIC—Lawrence Schweitzer, M.D., 23 Woodward Crescent, Halifax, Nova Scotia B3M1J6. Louvain 1975/Dalhousie 1979. Board eligible. Group, partnership, solo. Available July 1979.

T. Ghavamian, M.D., P.O. Box 3452, San Diego, CA 92103. Teheran 1953. Board certified. Group, partnership, solo, academic. Available.

SURGERY, THORACIC—Chukuma I. Okadigwe, M.D., 590 Flatbush Avenue, Apt. 14-P, Brooklyn, NY 11225. Colorado 1968. Board eligible. Group, partnership, solo. Available.

UROLOGY—Arnold M. Grebler, M.D., 19 Spartan Avenue, Staten Island, NY 10202. Bologna (Italy) 1974. Partnership or group. Available July 1979.

Stewart L. Feldman, M.D., 34 Hunter Circle, Mount Holly 08060. Vermont 1971. Board eligible (general surgery). Group, partnership. Available July 1979.

Ekiong C. Tan, M.D., 8478 Ohern Street, Omaha, Nebraska 68127. Santo Tomas (Philippines) 1970. Board eligible. Group or partnership. Available July 1979.

Daniel H. Piazza, M.D., 741 Peninsula Court, Ann Arbor, Michigan 48105. Illinois 1972. Board eligible. Partnership, single or multi-specialty group. Available July 1979.

Gerald A. Goldman, M.D., 291 Willowwood Drive, Erie, PA 16500. Guadalajara 1972. Board eligible. Association leading to partnership. Available July 1979.

Standards for Testing and Diagnostic Centers

Board of Medical Examiners' Rule N.J.A.C. 13:35-6.14

Edwin H. Albano, President of the Board of Medical Examiners in the Division of Consumer Affairs of the Department of Law and Public Safety, pursuant to authority of N.J.S.A. 45:9-1, *et seq.*, hereby adopts the following new regulation concerning Health Testing Centers and Diagnostic Centers or Clinics.

The performance of physical examinations accompanying diagnostic testing procedures on human beings is included within the practice of medicine. Such services presently are being offered to the public in both stationary and mobile facilities which are not in all circumstances regulated by the Department of Health. It is essential for a meaningful interpretation of test data that the underlying tests be administered competently and appropriately as determined and supervised by a licensed physician.

The purpose of this regulation is to define and establish minimum medical standards of operation for those centers, clinics, or facilities which provide or purport to provide activities such as physical examinations and/or laboratory testing procedures, which presently are regulated by N.J.S.A. 45:9-1 *et seq.* and 45:9-42.1 *et seq.*

The full text of the adopted regulation follows:

13:35-6.14 Standards concerning testing and diagnostic centers

(a) The provisions of this regulation shall be applicable to centers, clinics, or facilities which provide or offer to provide physical examinations and/or testing procedures.

(b) Definitions include the following:

1. "Automated Health Testing Center" (AHTC) means a facility under the control, supervision, direction, and physical presence of a licensed physician or a group of licensed physicians offering services to the medical profession or to the public, which screens patients by multiphasic methods, such screening resulting in a determination less complete than a physical examination performed by a licensed physician; and which does not purport to substitute for such complete physical examination. This definition is not intended to include a one-modality service, such as community chest x-ray examination, and community blood pressure examination.

2. "Diagnostic Center" or "Diagnostic Clinic" (hereafter "Center") means a facility under the control, supervision, direction, and physical presence of a licensed physician, which is staffed by one or more licensed physicians and which contains the facilities necessary to render a medical service by establishing a diagnosis and which may recommend a course of treatment for the patient.

3. "Licensed physician" means a duly licensed physician of the State of New Jersey who possesses a current unrestricted license to practice medicine and surgery.

(c) The licensed physician in charge of and responsible for the supervision and direction of the AHTC or the Center shall, prior to operation of the facility, submit for approval of the Board of Medical Examiners a copy of the licensing application previously approved by the Department of Health, proof of current licensure of physician in charge, and, if applicable, facility license pursuant to N.J.S.A. 45:9-42.26.

(d) The licensed physician in charge and the AHTC or Center shall have the following responsibilities:

1. Procure from the patient a designated physician or, in the absence of a designated physician, the addressee to whom all reports shall be directed.

2. Submit the full test report to the referring or designated physician and to the patient or designated addressee as instructed by the patient, not later than three business days from the date of receipt of such test report by the AHTC or Center.

3. Clearly identify all abnormalities for the attention of the physician.

4. Where abnormality is apparent, the notification to the patient shall be accompanied by an express recommendation that the patient contact the appropriate physician for clarification.

(e) Noncompliance with the above regulations may be considered gross malpractice or gross neglect by the licensee and may result in the institution of suspension or revocation proceedings.

(f) This rule shall be effective ninety (90) days after filing. (Filing date was December 14, 1978)

PERSONAL ITEMS

David Eckstein, M.D.

David Eckstein, who appears in our cover reproduction of the dust jacket of *The Geriatric Patient* (see review, page 233, this issue), was born in Trenton, and educated at Lehigh University, Jefferson Medical College, St. Francis Hospital in Trenton, and the City Hospital in New York. He was honored with membership in Phi Beta Kappa and Alpha Omega Alpha. Dave subsequently enjoyed a very successful practice in internal medicine in the city of his birth. Furthermore, he combined his clinical practice with careers as assistant pathologist at St. Francis Hospital and Assistant Medical Examiner for Mercer County between the years 1946 and 1956.

His devotion to organized medicine ultimately led him to the top of the ladder—chairmanship of the Board of Trustees of the Medical Society of New Jersey. During all these years, Dr. Eckstein gradually evolved a commitment to a special branch of internal medicine—geriatrics. In 1968 he joined the medical staff of the Meadow Lakes Retirement Community, Hightstown, and in 1970 became medical director of that institution.

Dave's love for his geriatric practice has been a pleasure to behold. More than that, he insists on sharing his enthusiasm and knowledge with anyone who will listen to him. This naturally led to chairmanship of the Joint Council To Improve Health Care of the Aged in New Jersey, membership on the AMA Committee on Aging, and AMA Delegate to the White House Conference on Aging in 1971. He directed meetings on "The Medical Director in the Long-Term Care Facility" for the AMA in 1975 and 1978.

David Eckstein has combined a career in medicine, which has gained national attention, with a genuine concern for his community and its people. Even now, he is busy helping to raise funds for new construction at the Greenwood House, a home for the aged Jewish residents of Trenton. Dave has made and continues to make significant contributions to his fellowman and woman of all ages.

William G. Wilkerson, M.D.

Announcement has been made of the appointment of William G. Wilkerson, M.D., as Medical Director of the Division of Disability Determinations of the Department of Labor and Industry, to succeed Eric H. Wolf, M.D., who retired on February 1, 1979. Dr. Wilkerson has been on the staff and Assistant Medical Director of this organization for the last five years.

He is well known throughout the medical community and the State, previously having been Director of the Drug Addiction and Alcohol Program for the State of New Jersey and chairman of the Rehabilitation Commission of the Goodwill Industries of America. In this position he was helpful in drafting the bill now pending before Congress concerning rehabilitation of patients who have medically documented impairments. He has served as president of the Hudson County Mental Health Association, chairman of the Board of Trustees of the Meadowview Hospital (Hudson Psychiatric Hospital), president for ten years of the Jersey City Chapter of the NAACP, and Vice President of the State Conference.

Dr. Wilkerson also was active in civic affairs, serving as Vice Chairman of the Housing Authority of Jersey City, Vice Chairman of the Board of Mental Health, and member of the National Board of Goodwill Industries.

In 1978, he was the recipient of the National Brotherhood award of the National Conference of Christians and Jews, and has been elected to a fellowship in the Royal Medical Society of London. He also was awarded an honorary doctorate degree by St. Peter's College.

FOR OCCUPATIONAL PHYSICIANS

A meeting on Employment of Disabled Citizens will be held on April 4, 1979, at Rutgers University Student Center in Newark. Agenda will include a workshop for physicians. Employment of the Handicapped under Section 503 of the Rehabilitation Act will be stressed.

FOR INFORMATION: Daniel J. O'Regan, M.D., Medical Director, New Jersey Division of Vocational Rehabilitation Services, Labor and Industry Building, John Fitch Plaza, Trenton, 08620, (609) 292-5987.

the physician and sportsmedicine

COLLEGE OF MEDICINE AND DENTISTRY
OF NEW JERSEY

Baseball Injuries—Prevention and Management
A Continuing Education Conference
March 29, 1979, 8:00 AM—5:30 PM

Presented by The Physician and Sportsmedicine and CMDNJ—New Jersey Medical School at the College's Medical Science Building, New Jersey Medical School, Newark, NJ

Speakers include the team physicians for The New York Yankees, Boston Red Sox, and Philadelphia Phillies, the President of Little League Baseball and other noted Sports Medicine specialists.

Opportunities for CME credits will be available.

For further information contact
CMDNJ—Department of Continuing Education
Box 101, University Heights
Piscataway, New Jersey 08854
Call 201/463-4707

ASSOCIATION FOR MEDICAL EDUCATION OF NEW JERSEY
in cooperation with
The Academy of Medicine of New Jersey
The Medical Society of New Jersey
and
The New Jersey Hospital Association
present a Symposium on

MEDICAL STAFF OFFICERS—THEIR GOALS AND ROLES

A discussion of the issues and problems facing community hospital medical staff officers with suggestions and recommendations for effective solutions.

WEDNESDAY, APRIL 4, 1979
at
Center for Health Affairs, Princeton, New Jersey
8:30 A.M.-4:00 P.M.

Registration: $15.00 (includes lunch)
Moderator: Arthur Bernstein, M.D.
Program Chairman: Brewster Miller, M.D.
Among the distinguished guests:
John E. Affeldt, M.D.
President, JCAH

For further information contact: AHME OF NEW JERSEY
2424 Morris Avenue
Union, New Jersey 07083
201-687-8780

CME CALENDAR

This listing is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, the Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the College of Medicine and Dentistry of New Jersey. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

ANESTHESIOLOGY

April

4 Inhalation Anesthesia
7-9 p.m.—Landwehr's Restaurant, River Rd., Trenton
(Mercer Medical Center and AMNJ)

MEDICINE (includes Family, Internal, and General Medicine and Dermatology)

April

3 Everyday Problems in the Newborn Nursery

10 Acute Myocardial Infarction Patients
8-9 a.m.—Garden State Community Hospital, Route 73, Marlton
(Garden State Community Hospital and AMNJ)

4 Congenital Heart in Adults
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

4 Medical Grand Rounds
11:30 a.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

4 Patho-Physiology of Infarction

11 Management in the Coronary Care Unit

18 Sudden Death in Coronary Heart Disease

25 Management of Heart Failure
10 a.m.-12 noon—St. Michael's Medical Center, Newark
(St. Michael's Medical Center and AMNJ)

4 Lung Disorders and Blood Gases
10:30-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

4 Nonmalignant Disorders of the Bowel

11 Fever of Unknown Origin

18 Contact Dermatitis 1979
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

4 Dinner Meeting
6:30-9:30—Holiday Inn, East Orange
(AMNJ Endocrinology Section)

4 Nutrition-Elemental Diets

25 Medical Lecture Series
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

4 Endocrine Conferences
11 3:30-5 p.m.—Rotates between Newark
18 Beth Israel Medical Center, Martland
25 Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

4 Advances in Medicine
11 9:30-11 a.m.—Bergen Pines County
18 Hospital, Paramus
25 *(Bergen Pines County Hospital and AMNJ)*

5 Cardiology Conferences
12 2:15-4:15 p.m.—Deborah Heart and
19 Lung Center, Browns Mills
26 *(Deborah Heart and Lung Center and AMNJ)*

6 Arthritis
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

6 Acute Respiratory Failure

13 Bronchial Asthma Update

20 COPD and Rehabilitation

27 O_2 Therapy
11:45 a.m.-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Hospital and AMNJ)

7 Endocrine Disorders Including Diabetes Mellitus
9 a.m.-5 p.m.—Stevens Institute of Technology, Hoboken
(Hudson County Medical Society and AMNJ)

10 Sudden Death in Young Athletes
12:45-2 p.m.—Mercer Medical Center, Trenton
(American Heart Association, Mercer County Chapter)

10 Endocrinology and Metabolism Testing
9 p.m.—West Hudson Hospital, Kearny
(West Hudson Hospital and AMNJ)

10 Immunology
8-10 p.m.—Schering Corporation, Kenilworth
(NJ Dermatological Society and AMNJ)

12 Case Presentations and Guest Speakers
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

12 Virology
19 4-6 p.m.—Institute for Medical Research,
26 Copewood St., Camden
(Institute for Medical Research and AMNJ)

18 Thromboembolic Disease
11:30 a.m.-1 p.m.—VA Hospital, East Orange
(VA Hospital and AMNJ)

19 Regional Chest Conferences
7:30-9:30 p.m.—St. Joseph's Medical Center, Paterson
(NJ Thoracic Society and AMNJ)

19 Juvenile Diabetes
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

23-24 Combined Health Care Professional Course
8:30 a.m.-4:30 p.m. each day—Meadowlands Hilton Hotel, Secaucus
(American Diabetes Association and AMNJ)

24 Upper Gastrointestinal Bleeding
8-9 a.m.—Greater Paterson General Hospital, Wayne
(Greater Paterson General Hospital and AMNJ)

24 Lower Back Pain
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

25 Cardiac Grand Rounds
3-4:30 p.m.—NJ Medical School, Newark
(American Heart Association, NJ Affiliate, and AMNJ)

25 William P. Burpeau Award Dinner
6:30 p.m.—The Manor, West Orange
(Urology Section of AMNJ)

May

1 Outpatient Procedures

29 Spondyloarthropathy
8-9 a.m.—Garden State Community Hospital, Route 73, Marlton
(Garden State Community Hospital and AMNJ)

2 Allergy: Asthma

9 Acute Renal Failure
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

8 Gram Negative Sepsis
8:30-9:30 p.m.—Riverside General Hospital, Secaucus
(Riverside General Hospital and AMNJ)

9 Surgically Corrected Congenital Heart Disease
12:45-2 p.m.—St. Francis Medical Center, Trenton
(American Heart Association and AMNJ)

10 Regional Chest Conferences
7:30-9:30 p.m.—The Valley Hospital, Ridgewood
(NJ Thoracic Society and AMNJ)

19-20 Recent Advances in Laboratory Medicine
9 a.m.-4:30 p.m.—Metpath Institute for Medical Education, 60 Commerce Way, Hackensack
(Metpath Institute for Medical Education and AMNJ)

23 Physical Medicine and Rehabilitation for the Family Practitioner
8:30 a.m.-1 p.m.—John F. Kennedy Medi-

cal Center, Edison
(AMNJ)

NEUROLOGY/PSYCHIATRY

April

2 **Voyeurism and Sexual Deviation**
8-10 p.m.—60 Melrose Place, Montclair
(Essex Psychiatric Seminar and AMNJ)

2 **Neuroscience Conferences**
9 11:30 a.m.-12:30 p.m.—Bergen Pines
16 County Hospital, Paramus
23 *(Bergen Pines County Hospital and*
30 *(AMNJ)*

3 **Seminar on Law and Psychiatry**
10 3:30-5:30 p.m.—Rutgers Law School,
17 Newark
24 *(Rutgers University Law School and AMNJ)*

4 **Infant Day Care**
7:30-9:30 p.m.—Marriott Motor Inn, Saddle Brook
(NJ Psychiatric Association, NJ Council of Child and Adolescent Psychiatry and AMNJ)

4 **Delayed Grief Reactions**

18 **Hyperactive Children**
1:30-3 p.m.—NJ Medical School, Newark
(NJ Medical School, Dept. of Psychiatry)

4 **Violence-Evaluation/Concepts**

18 **Psychological Testing**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

4 **Course for Psychiatrists—Semester II**
11 8-10 p.m.—Hackensack Hospital
18 *(NJ Psychoanalytic Society and AMNJ)*

5 **Psychotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, South Orange
(Group for Advanced Psychiatric Study and AMNJ)

5 **Psychiatric Lecture Series**
12 11 a.m.-12 noon—Greystone Park
19 Psychiatric Hospital
26 *(Greystone Park Psychiatric Hospital and AMNJ)*

5 **Schizophrenic Syndrome**

12 **Psychoneuroendocrinology Psychiatry**

19 **Cerebral Dysrhythmia and Schizophrenia**

26 12 noon-1 p.m.—Carrier Foundation, Belle Mead
(Carrier Foundation)

6 **Psychiatric Lecture Series**
20 1:30-5 p.m.—Trenton Psychiatric
27 Hospital
(Trenton Psychiatric Hospital and AMNJ)

7 **Pediatric Neurology**
Morristown Memorial Hospital
(Morristown Memorial Hospital and AMNJ)

10 **Cerebral Vascular Disease**

17 **Headache**

24 **Seizure Disorders**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

11 **Psycho-Sexual Dysfunctions in Clinical Practice**
8-9 a.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

11 **Sleep Disorders**
8:30-10:30 p.m.—Guido's Restaurant, Hackensack
(North Jersey Psychiatric Society and AMNJ)

11 **Schizophrenia in a State Hospital**
1:15-2:45 p.m.—Marlboro Psychiatric Hospital
(Marlboro Psychiatric Hospital and AMNJ)

11 **Depression and Anxiety**
1:30-2:30 p.m.—John E. Runnells Hospital of Berkeley Heights
(John E. Runnells Hospital and AMNJ)

18 **Alcoholism**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

May

2 **Personality Disorders**
11:30 a.m.-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

9 **Suicidology**
1:30 p.m.—John E. Runnells Hospital, Berkeley Heights
(John E. Runnells Hospital and AMNJ)

OBSTETRICS/GYNECOLOGY

April

4 **Lectures in Obstetrics/Gynecology**
8-10 p.m.—Location varies
(CMDNJ and AMNJ)

4 **Grand Rounds and Case Presentations**
11 2-4 p.m.—Rotating between Martland
18 Medical Center, Newark Beth Israel,
25 St. Michael's, St. Joseph's Hospitals, and Jersey City Medical Center
(CMDNJ and AMNJ)

5 **Grand Rounds and Case Presentations**
12 4-5 p.m.—Martland Hospital,
19 Newark
26 *(CMDNJ and AMNJ)*

11 **Infertility**
11:45 a.m.-12:45 p.m.—John F. Kennedy Hospital, Edison
(John F. Kennedy Hospital and AMNJ)

PATHOLOGY/CLINICAL PATHOLOGY

April

4 **Clinical Pathology Grand Rounds**
11 12 noon-1 p.m.—NJ Medical School,
18 Newark
25 *(CMDNJ and AMNJ)*

25 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School
(CMDNJ and AMNJ)

25- **Aspiration Cytology**
27 All day—Sheraton-Deauville, Atlantic City
(Hahnemann Medical College and Atlantic City Medical Center)

PEDIATRICS

April

6- **Spring Meeting-Nutrition**
8 Royal Sonesta Hotel, New Orleans
(NJ Chapter, American Academy of Pediatrics and AMNJ)

6 **Advances in Pediatrics**
20 9:30-10:30 a.m.—NJ Medical School,
27 Newark
(CMDNJ and AMNJ)

7 **Pediatric Neurology**
9 a.m.-4:30 p.m.—Morristown Memorial Hospital
(Morristown Memorial and AMNJ)

18 **Pediatric Dentistry**
8 a.m.-3:30 p.m.—VA Hospital, East Orange
(AMNJ)

19 **Juvenile Diabetes**
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

May

24 **Pediatric and Adolescent Gynecology**
8:30-10 p.m.—179 So. Maple Ave., Ridgewood
(American Medical Women's Association and AMNJ)

RADIOLOGY

April

19 **Ultrasound**
7:15-10:15 p.m.—Hospital Center at Orange
(Diagnostic Radiology Section for Northern NJ and AMNJ)

24 **Integrated Body Imaging**
8-9 a.m.—Garden State Community Hospital, Route 73, Marlton
(Garden State Community Hospital and AMNJ)

May

8 **Upper Extremity Trauma**
8-9 a.m.—Garden State Community Hospital, Route 73, Marlton
(Garden State Community Hospital and AMNJ)

16 **Advances in X-Ray Diagnosis and Treatment**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

16 **Dinner Meeting**
6:30 p.m.—The Manor, West Orange
(Radiotherapy Section, AMNJ)

GENERAL SURGERY

April

2 **Lecture Series in Surgery**
9 4:30-5:30 p.m.—NJ Medical School,
16 Newark
23 *(CMDNJ and AMNJ)*
30

3 **Weekly Tumor Conferences**
10 11 a.m.-12 noon—Morristown
17 Memorial Hospital
24 *(Morristown Memorial Hospital and AMNJ)*

17 **Colon and Rectal Cancer**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

21 **Cancer Seminar**
8:30 a.m.-12:30 p.m.—Ramada Inn, New Brunswick
(Philippine-American Medical Society of NJ and AMNJ)

25 **Colorectal Polyps and Cancers**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

May

1 **Weekly Tumor Conferences**
8 11 a.m.-12 noon—Morristown
15 Memorial Hospital
22 *(Morristown Memorial Hospital*
29 *and AMNJ)*

SURGICAL SPECIALTIES (includes ENT, neurosurgery, ophthalmology, orthopedics, plastics, and vascular surgery)

April

5 Neurosurgical Case Presentations
12 4-5:30 p.m.—NJ Medical School,
19 Newark
(CMDNJ and AMNJ)

6 Orthopedics
13 7:30-9 a.m.—Alexian Brothers Hospital,
27 Elizabeth
(Alexian Brothers Hospital)

19 Diseases of the Skeletomuscular System
8-10 p.m.—NJ Medical School, Newark
(Essex County Medical Society and AMNJ)

24 Lower Back Pain
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

24 Hemi-Orthoplasty in Acute Femoral Neck Fractures
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

May

22 Plastic Surgery in Salvaging Lower Extremity
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

MISCELLANEOUS

April

3 Malpractice
8-9 p.m.—Burdette Tomlin Memorial Hospital, Cape May Court House
(Burdette Tomlin Memorial Hospital and AMNJ)

4 Clinical Pharmacology
11:30 a.m.-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

18 Dental Symposium
(All Day) VA Hospital, East Orange
(VA Hospital and AMNJ)

May

3 Medical Aspects of Baseball
All day symposium—NJ Medical School, Newark
(CMDNJ)

9 Suicidology
1:30 p.m.—John E. Runnells Hospital, Berkeley Heights
(John E. Runnells Hospital and AMNJ)

9 Annual Awards Dinner
6 p.m.—Chanticler, Millburn
(AMNJ)

13 Annual Meeting
8 a.m.-12 noon—Atlantic City
(Oncology Society of NJ and AMNJ)

See Following Page For Additional CME Opportunities

THE DEPARTMENT OF MEDICINE and THE PAGE AND WILLIAM BLACK POST-GRADUATE SCHOOL OF MEDICINE of the MOUNT SINAI SCHOOL OF MEDICINE (CUNY)

Announce a Postgraduate Course

New Approaches to Old Problems in Internal Medicine

Under the Direction of
RICHARD GORLIN, M.D. HARRIET S. GILBERT, M.D.
and FACULTY of the MOUNT SINAI SCHOOL OF MEDICINE

May 15-18, 1979

Tuesday-Thursday, 9:00 AM to 5:00 PM; Friday, 9:00 AM to 4:00 PM, (4 Sessions)

The practice of Internal Medicine presents a continuing challenge to integrate new concepts of diagnosis and therapy into the context of patient management. This four-day multidisciplinary course in Internal Medicine is designed for the practicing physician and will cut across subspecialties to address commonly encountered clinical situations by means of concise lectures, panel discussions and workshops. Each session will be problem-oriented and based on illustrative case material. Opportunity will be provided for registrants to clarify questions arising from their own clinical experience through question-and-answer periods and informal discussions with faculty.

One afternoon will be devoted to participation in small subspecialty workshops or may be spent with members of the faculty who will be available by advance arrangement to discuss topics of particular interest to the registrants. This session will be followed by attendance at Medical Grand Rounds.

A syllabus summarizing the questions to be addressed by each speaker, pertinent clinical material, and references will be distributed.

FEE: $300.00 (including Luncheon and Cocktail Party)

This course will be given at the Mount Sinai Medical Center, New York, New York

APPLY TO: Director, The Page and William Black Post-Graduate School of Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, New York 10029.
Tel: (212) 650-6737.

JEFFERSON MEDICAL COLLEGE OF THOMAS JEFFERSON UNIVERSITY

Presents

MODERN THERAPEUTICS V

March 28-30, 1979

Fee: $165

AMA Accredited

For further information contact:

Office of Continuing Medical Education
Jefferson Medical College
1025 Walnut Street
Philadelphia, Pa. 19107
(215) 928-6992

JEFFERSON MEDICAL COLLEGE OF THOMAS JEFFERSON UNIVERSITY

Department of Psychiatry and Human Behavior

Presents

THE SECOND ANNUAL CONFERENCE AND WORKSHOP ON PSYCHOSOMATIC DISORDERS

April 26, 27, 28, 1979

MEDICINE LOOKS AT STRESS

For Further Information Contact:

Office of Continuing Medical Education
Jefferson Medical College
1025 Walnut Street
Philadelphia, Pa. 19107
(215) 928-6992

AMA Accredited

REHABILITATION OF PATIENTS WITH STROKE OR HIP FRACTURE

Presented by
University of Maryland
School of Medicine

This course is designed for physicians who care for disabled patients in the home, in a nursing home, or in an extended care facility. It will feature discussions and clinical presentations which are relevant to the current medical and surgical management of stroke and of various kinds of hip fractures. The clinical presentations will feature "live patients" undergoing active rehabilitation.

DATES: May 11 - 12, 1979

LOCATION: John L. Deaton Medical Center
Baltimore, Maryland

For additional information:

CONTACT: Program of Continuing Education
University of Maryland
School of Medicine
10 S. Pine Street
Baltimore, Maryland 21201
(301) 528-3956

PROBLEMS IN EMBRYONIC & EARLY FETAL DEVELOPMENT

Presented by
The University of Maryland
School of Medicine
Department of Obstetrics & Gynecology
and
Program of Continuing Education

This course will review some important principles of early fetal development. Recent developments in the methods of diagnosis and management of problems affecting the fetus during this early stage will be presented so that clinicians will be able to recognize common problems that may affect the fetus, understand their pathophysiology, and plan an organized approach to the management of such conditions.

DATES: May 18 and 19, 1979

LOCATION: The Baltimore Hilton Inn
Baltimore, Maryland

For additional information:

CONTACT: Program of Continuing Education
University of Maryland
School of Medicine
10 S. Pine Street
Baltimore, Maryland 21201
(301) 528-3956

OBITUARIES

Dr. Albert G. K. Andrews

On January 4, Albert George K. Andrews, M.D., a member of our Passaic County component, died in Passaic Beth Israel Hospital. A native of the West Indies, Dr. Andrews was graduated from Howard University Medical School in 1928 and had practiced general medicine in the Passaic area since 1930. He had been on the staff at Beth Israel, Passaic General, and St. Mary's Hospitals in Passaic and Barnert Memorial Hospital in Paterson.

Dr. Sigurd W. Johnsen

Sigurd W. Johnsen, M.D., the 159th President of the Medical Society of New Jersey (1951-1952), died on December 26, 1978, at the age of 83. A native of Chicago, Dr. Johnsen earned his medical degree from Cornell University Medical College in 1925 and pursued graduate work in gastroenterology and radiology at New York Postgraduate Medical School and Columbia University. He was a Fellow of the American College of Physicians, the Radiological Society of North America, and the American College of Gastroenterology, of which he served a term as president. The latter had been formed from the National Gastroenterological Association of which Dr. Johnsen was a Fellow, member of the executive board, and secretary. He also had been elected to Fellowship in the International Gastroenterological Association. A member of our Passaic County component, he had maintained a practice in the City of Passaic from 1926 until retirement in 1971. He was affiliated with the Passaic General Hospital as senior attending gastroenterologist and served as a member of the medical board from 1947 to 1951 and as chief of staff from 1949 to 1951. Dr. Johnsen had been actively engaged in Medical Society affairs since the late 1930s as a member and chairman of several committees and scientific sections, and was elected to the Board of Trustees in 1946. Among his survivors is a son, Dr. Sigurd E. Johnsen, a practicing radiologist in Passaic County.

Dr. Fred Sachs

On December 2, 1978, Fred Sachs, M.D., of Jersey City, died at his home. Born in 1904 and graduated from the University of Michigan School of Medicine in 1927, Dr. Sachs pursued a residency in general surgery before returning to New Jersey to establish a practice in that field. A member of the Hudson County Medical Society, he had been associated with the Medical Center and Greenville Hospital in Jersey City. Dr. Sachs was a Fellow of the International College of Surgeons, and during World War II served in the medical department of the Army of the United States.

Dr. Irving Shavelson

On January 3, Irving C. Shavelson, M.D., formerly of Atlantic City, died in Hollywood, Florida. Born in New York City in 1905, Dr. Shavelson earned his medical degree from the Long Island College of Medicine in 1928. He was board certified in internal medicine and practiced cardiology in Atlantic City for many years. He had been chief of that department at the Atlantic City Medical Center. Dr. Shavelson had been semi-retired since 1964, working only part-time during the summer months in Atlantic City. He was a Fellow of the American College of Physicians and an organizer and first president of the Atlantic County Heart Association. During World War II he served in the department of medicine of the Army of the United States.

Dr. Jacob Stark

Jacob Stark, M.D., a member of the Passaic County component, died on January 18 at Barnert Memorial Hospital. Dr. Stark, a native of Russia, born in 1904, was educated in the United States and was a graduate of Boston University's medical school, class of 1928. He practiced family medicine in the Paterson area for 50 years, and had been affiliated with Barnert Memorial Hospital.

Dr. Israel J. Wolf

Israel J. Wolf, M.D., formerly of Paterson, died on January 3, after a prolonged illness. Born in Connecticut in 1903, Dr. Wolf was graduated from McGill University Medical School (Canada) in 1928 and took graduate work at the University of Vienna. He practiced pediatrics with special interest in pediatric allergy in Paterson for many years. Dr. Wolf was a diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics and of the American College of Allergy. He had been on the staff at Barnert Memorial Hospital in Paterson, where he had served a term on the medical board, and had been on the faculty at Albert Einstein College of Medicine in New York.

Housing Application

213th Annual Meeting

The Medical Society of New Jersey

May 12-15, 1979

	DAILY RATES		
	Single	Twin	Suites
Holiday Inn	$42	$46	Rates quoted upon
(MSNJ Headquarters)	50	54	request
	58	62	
Howard Johnson's	$42	$46	Rates quoted upon
(Auxiliary Headquarters)	50	54	request
	58	62	
Resorts International	Rates Upon Request		

If room is not available at rate requested, next available rate will be assigned

Mail this application directly to the Atlantic City Convention Bureau
16 Central Pier
Atlantic City, N.J. 08401

Please list 1st and 2nd choice; confirmation will come directly from hotel.

1st Choice.....2nd Choice.....

Accommodations desired: ☐ Single ☐ Twin ☐ Suite Parlor & 1 Bedroom
☐ Suite Parlor & 2 Bedrooms

Name.....

Address.....

City..... State..... Zip.....

Phone.....

Will arrive..... (DATE) Time..... Will depart..... (DATE) Time.....

☐ Check if Official Delegate..... County

BOOK REVIEWS

The Geriatric Patient

William Reichel, M.D., Editor. New York, H.P. Publishing Company, 1978. Pp. 233. Illustrated. Price not given.

William Reichel, Chairman of the Department of Family Practice at the Franklin Square Hospital and 26 contributing authors have compiled a wealth of information—didactic, epidemiologic, and pragmatic—in a book which should be of interest to every primary physician. Based on the premise that "the care of the elderly is overwhelmingly in the domain of primary practice," this compilation does, indeed, represent a handbook which touches on virtually every major consideration of this age group.

The authors have included numerous golden nuggets culled from their own experiences which can make us more comfortable in our assessment of the elderly and our patients more comfortable as a result of our treatment. Some examples follow:

"The physician should ask that the prescription label instructions be in large type and they should be crystal clear: 'Take one pill with a full glass of water at breakfast.' " *Lamy and Vestal*

"Old people do not complain unnecessarily. Their complaints may not be a reasonable interpretation of what is wrong. It's up to the physician to find out. Most of the time, we find that the elderly underplay their symptoms and problems." *Eckstein*

"The presumption of senility" or "chronic brain syndrome" is unwarranted in the context of sudden behavioral change; rather, we may be dealing with the behavioral concomitants of a reversible medical illness or drug toxicity." *Reichel*

"Rehabilitation means an attempt to return a person, insofar as possible, to a life-style the patient used to have, not the creation of a new one." *Joffe*

The 25 chapters of this book contain many tables and algorithms of practical value to the reader. The abundant use of x-ray reproductions and other special studies, including echocardiograms, photographs, and artistic representations, enhance the opportunity of the reader to learn. References are selected but abundant.

The authors of each chapter are leaders in the study and practice of medicine and surgery as it applies to the elderly. One might summarize the message of this reference text in the words of Robert N. Butler, Pulitzer Prize winner and director of the National Institute on Aging:

"The key to geriatrics is comprehensiveness of evaluation, which also happens to be a key to improved physician-patient relationships."

I strongly urge each primary physician to obtain and read *The Geriatric Patient.* It will help to cleanse us of many myths and prejudices we all harbor.

A. Krosnick, M.D.

Between You and Me.

J. Parrish, M.D., B. Gilchrest, M.D., and Thomas Fitzpatrick, M.D. Boston, Little, Brown & Co., 1978. Pp. 239. ($8.95)

We are bombarded daily through every available means of communication by a great mass of misinformation and pseudo facts about skin health and skin care—much of it deliberately misleading. There is an urgent need, therefore, for dermatologists to inform the laity, which in this context includes also their fellow physicians.

I think that this book does not perform its stated function—for the general public, at least. The terse, graphic messages of the media cannot be countered effectively by the often turgid prose which ranges in content from simplistic naiveté to simple pretentiousness. I am left with the fervent hope that these most eminent authors had the help of a ghost who can be blamed for most of these journalistic efflorescences.

The book begins with a philosophical essay on beauty. This is followed by what I presume to be the authors' view of psychology—all rather quaint. Life is portrayed as a struggle between the "human" outside and the "animal" inside—a sort of Victorian version of "Beauty and the Beast."

Happily after that, the authors get down to what they really know. To my great joy, I find that they know exactly what I know, and many of my colleagues in the field might enjoy this book for the same reason. Any non-dermatologic physicians in possession of the simple facts enumerated in this work should be able to handle expertly the great bulk of the dermatologic problems they might encounter in their practice. For the laity, however, there are too many, too specialized words, and no simple line drawings.

Peter J. Koblenzer, M.D.

Antibiotics Indications Device

Excerpta Medica Services, Inc., Princeton, NJ, 1978. Pp 32. (No price given)

Dr. Max Findland, the great leader of our infectious disease world, has put together a fine editorial staff for Excerpta Medica Services for an *Antibiotics Indications Device.* This device consists of a slide of organisms and antibiotics with the recommended dosages and a booklet which contains additional information on specific infections, less frequent infections, toxicity of antibiotics, and a superb bibliography. Overall, a great deal of work has gone into this very compact booklet and slide rule. On initial examination the attractiveness of the package makes the effort worthwhile. However, on closer examination, the density of the material overwhelms the user. First of all physicians, unlike engineers, don't like slide rules. Secondly, the tables are unimaginative and monotonous. The contents are good but the method used, I would venture to say, will not have much appeal for the busy practitioner. The telephone hot-line service, computers, and textbooks have more appeal. In all, the attempt is honorable, but the method is outdated.

Leon G. Smith, M.D.

THE JOURNAL OF THE MEDICAL SOCIETY OF NEW JERSEY

Publisher: The Medical Society of New Jersey
Two Princess Road
Lawrenceville, N.J. 08648

Advertising Representative: United Media Associates
16 Bruce Park Avenue
Greenwich, Conn. 06830
(203) 661-9702

General Information

1. Issuance:

a. Frequency: Monthly

b. Issue date: 10th of month.

c. Mailing date: 10th of month.

2. Established: 1904

3. Organization Affiliation:

Official publication of
The Medical Society of New Jersey.

4. Circulation Data:

a. Controlled circulation to all members of the Medical Society of New Jersey. Members' subscription ($5) is included in Society dues. Rates for nonmembers $10, outside USA add $4.00 for postage. Single copies $1.

b. Annual percentage of subscription renewals: 100% of members.

c. Number of issues sent after subscription expiration: None.

5. Special Issues: Convention
Annual Transactions
Index (December)

6. Editorial Content: Original scientific articles, special articles, case reports, editorials, medical news and meeting notices, trustees' minutes, communicable disease reports, state legislation, convention, medical insurance, PSRO, education, CME, etc.

7. Requirements for Acceptance of New Professional Products for Advertising.

All advertising subject to Publication Committee approval.

8. Requirement for Advertising Clearance:

All advertising subject to Publication Committee approval.

9. Advertising Acceptance of Nonprofessional Products or Services:

All advertising subject to Publication Committee approval

10. Policy on Placement of Advertising:

Advertising is interspersed throughout the publication. All R.O.B. ads are rotated each month. Advertising and editorial material is not placed on the same page.

11. Advertisers' Index: No.

12. Services to Advertisers:

a. Availability of mailing list: Availability subject to approval by Board of Trustees.

b. Availability of editorial reprints: Please direct such requests to the Editor.

13. Staff:

Editor: Arthur Krosnick, M.D. Associate Editors William A. Dwyer, Jr., M.D.; Bernard D. Pinck, M.D., Assistant Editor: Marjorie D. Treptow; Executive Director: Vincent A. Maressa.

14. Circulation:

All members of the Medical Society of New Jersey.

15. Guaranteed Circulation:

All members of the Medical Society of New Jersey.

16. Circulation Verification:

Publisher's statement, postal receipt verification.

17. Rates Per Thousand:

Based on the 12-times rate of $270 and circulation of 10,000: $27.00.

THE JOURNAL of The Medical Society of NEW JERSEY Rate Card effective January 1979

8. Coverage and Market

. Coverage: All members of The Medical Society of New Jersey, plus trade circulation. Circulation figures as of 6-1-78.

GP	2,790
IM	1,597
GS	1,222
OBG	693
PED	582
DERM	170
ALL	68
UROL	181
OTO	368
PSYCH	547
OPH	322
CARD	78
GASTRO	28
ANES	234
TOTAL	9,687

. Trade Circulation:
Non-member physicians
Medical libraries
Medical schools
Drug manufacturers
Medical book publishers
Medical abstract services
Advertisers
Advertising agencies
Subscriptions
Total approximately 650

l. Readership Survey: A readership Survey by Health Industries Research shows that the JOURNAL is extremely well read. Copies are available upon request.

19. MEMBERSHIP CIRCULATION BY COUNTIES OF STATE OF NEW JERSEY

Atlantic	240
Bergen	1,157
Burlington	285
Camden	567
Cape May	51
Cumberland	141
Essex	1,848
Gloucester	110
Hudson	594
Hunterdon	78
Mercer	599
Middlesex	578
Monmouth	589
Morris	522
Ocean	247
Passaic	763
Salem	52
Somerset	183
Sussex	74
Union	846
Warren	71
Total	9,595

Guaranteed Circulation: 10,000

THE JOURNAL of The Medical Society of NEW JERSEY Rate Card effective January 1979

Rates

20. Issuance:

a. Frequency: Monthly

b. Issue Date: 10th of month

c. Mailing date: 10th of month.

21. Closing Dates for Space:

a. Reservations: 1st of month preceding month of issue

b. Cancellations: 6th of month preceding month of issue

22. Agency Commission: 15%

23. Cash Discount: 2%, 10 days.

24. Rates:

	1 time	3 times	6 times	12 times
1 page	310	300	290	270
1/2 page	180	170	160	150
1/4 page	85	80	75	70
1/8 page	55	50	45	40

Classified: Available to member physicians only.

25. Earned Rates:

Rates based on number of insertions used within one year, regardless of size. Space purchased by a parent company and subsidiaries is combined for accounting of earned rates.

26. Color Rates:

a. Standard color $120 plus earned black and white rate.

b. List of standard colors: AAAA standard red, green, blue, yellow, orange.

c. Matched Colors: $130 plus earned black and white rate.

d. 3-color rate: $390 plus earned black and white rate.

e. 4-color rate: $440 plus earned black and white rate.

Special 4-color rates are available to advertisers placing insertions in the Five Key State Journals (Illinois, New Jersey, New York, Ohio and Pennsylvania). Contact UMA.

27. Bleed: 10%

28. Inserts:

60 pound coated only.

29. Preferred Position Rates:

a. Preferred position rates quoted on request and subject to availability.

b. Should an advertiser insist on a specific position, production requirements may dictate premium rates.

30. Miscellaneous:

a. Contract requirements: All contracts subject to publisher's approval.

b. Statement of guarantee of uniform rates and discounts to all advertisers using same amount and kind of space: No exceptions to published rates.

c. Concessions: None

d. Rates subject to change with 90 days notice. Contracts accepted with understanding that rates will be guaranteed for three months beyond last issue closed. In the event of rate increase, contracts may be terminated without penalty of short rate.

THE JOURNAL of The Medical Society of NEW JERSEY Rate Card effective January 1979

Mechanical Requirements

THE JOURNAL is printed by offset.
Trim size: 8 x 11

31. Plate Sizes:

Page Unit	Dimensions
1 full page	7 x 10
1/2 horizontal	7 x 4-7/8
1/2 vertical	3-3/8 x 10
1/4 horizontal	7 x 2-3/8
1/4 vertical	3-3/8 x 4-7/8
1/8 horizontal	7 x 1-1/8
1/8 vertical	3-3/8 x 2-3/8

32. Bleed Sizes:

Page Unit	Dimensions
1 full page	8-1/8 x 11-1/4
1/2 horizontal	8-1/8 x 5-5/8
1/2 vertical	11-1/4 x 4-1/16

33. Insert Requirements:
Untrimmed size—8-1/4 x 11-1/4
Quantity—11,500

34. Paper Stock: Covers: 80-pound.
Inside pages: 45 or 50 pound.

35. Type of Binding: Perfect bound.

36. Halftone Screen: Up to 133 screen.

37. Reproduction Requirements:
Black and white positives and 2-color advertisements: negatives, camera-ready mechanicals, and art work acceptable.

4-color: film negatives or positive separations and press proof.

Offset film negatives or positives on .002 or .004 stable base materials must have register marks, center marks, and trim marks clearly indicated. Each negative must be marked for color and be right reading emulsion side down.

38. Closing Dates:

a. Negatives or positives, camera ready mechanicals, and art work: 10th of the month preceding month of issue.

b. Publication set copy: 5th of month preceding month of issue.

39. Disposition of Reproduction Material:
Material is held for one year and then destroyed.

40. "Dual Responsibility": Billing directed to the advertising agency at the net rate is approved on condition that the advertiser will accept "dual responsibility" for payment if the agency does not remit within 90 days. Advertising accounts in arrears 60 days will be billed in duplicate to both the agency and the advertiser.

41. Addresses:

Sales, general information:
United Media Associates
16 Bruce Park Avenue
Greenwich, Connecticut 06830
203-661-9702

Contracts and insertion orders:
The Journal Medical Society of New Jersey
370 Morris Avenue
Trenton, N.J. 08611
609-393-7196

Reproduction material:
Journal of M.S.N.J.
Two Princess Road
Lawrenceville, N.J. 08648
609-393-7196

Inserts: (11,500)
Journal of M.S.N.J.
c/o Hughes Printing Company
34 North Crystal
East Stroudsburg, Pa. 18301

EMERGENCY DEPARTMENT PHYSICIAN

Full-time emergency physician in lovely shore community with active emergency department. Salary negotiable. Liberal benefits including malpractice insurance, BC/BS, major medical, retirement plan, and life insurance. Must be qualified by formal training and experience.

Apply: James M. Wilson, Assistant Administrator, Southern Ocean County Hospital, 1140 West Bay Avenue, Manahawkin, NJ 08050.

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Narthwestern New Jersey haspital seeks Board Certified Family Practice physician ta direct a develaping two-haspital shared family practice residency pragram. Pragram is ta be based in a satellite ambulatar facility nearby the smaller af the twa New Jersey haspitals and assaciated with a six-year ald high regarded family practice residency in the larger hospital which has a New Yark medical schaal affiliatian. Papulatian served primarily is rural. Lacatian is 1 1/2 haurs fram New York City and Philadelphia and adajacent ta recreational and educatianal centers. Teaching in an ambulatary facility and administrative experience an asset.

Contact: Family Practive Pragram/ % Administratar, Warren Haspital 185 Raseberry St., Phillipsburg, N.J. 08865

NEEMA EMERGENCY MEDICAL
—a professional association—

EMERGENCY MEDICINE POSITIONS: available with emergency physician group throughout Pa., N.Y., N.J., Mich. and Southeastern U.S. including all suburban, rural and metropolitan areas. Fee-for-service with minimum guarantee provided. Malpractice paid. Practice credits towards board certification. Physician department directors also desired. Please send resume to:

NEEMA Emergency Medical
Ste 400
399 Market St.
Phila., Pa. 19106

or phone
(215) 925-3511

Supervisor Physical Therapy

Major New Jersey Mental Health care facility seeking Board Licensed Therapist. Required 3 years experience in accredited hospital/clinic. Salary range $15,992 to $21,588. Excellent fringe benefits, easy access to New York City and Philadelphia.

Personnel Department
Trenton Psychiatric Hospital

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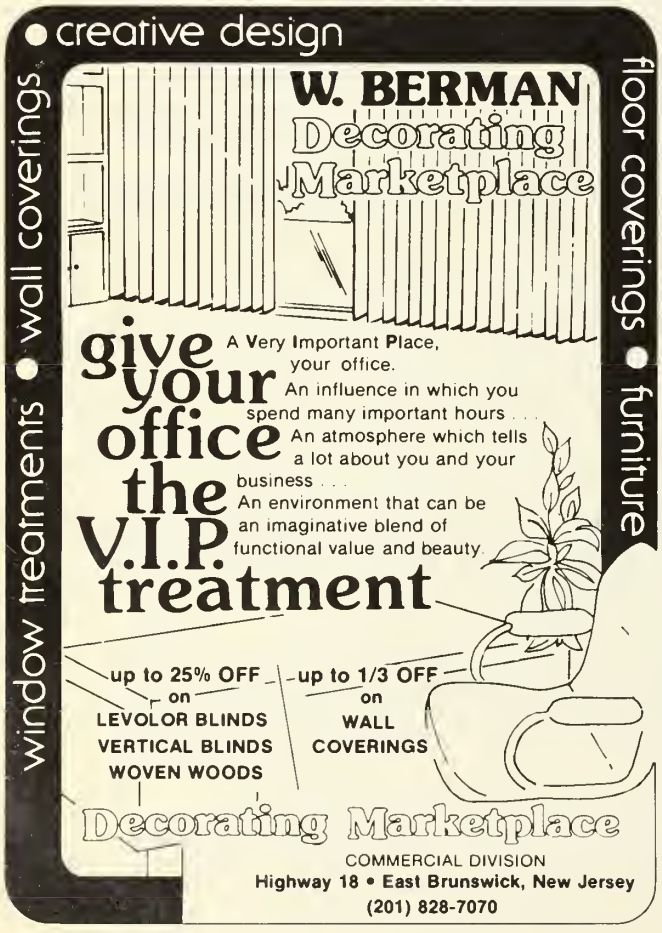
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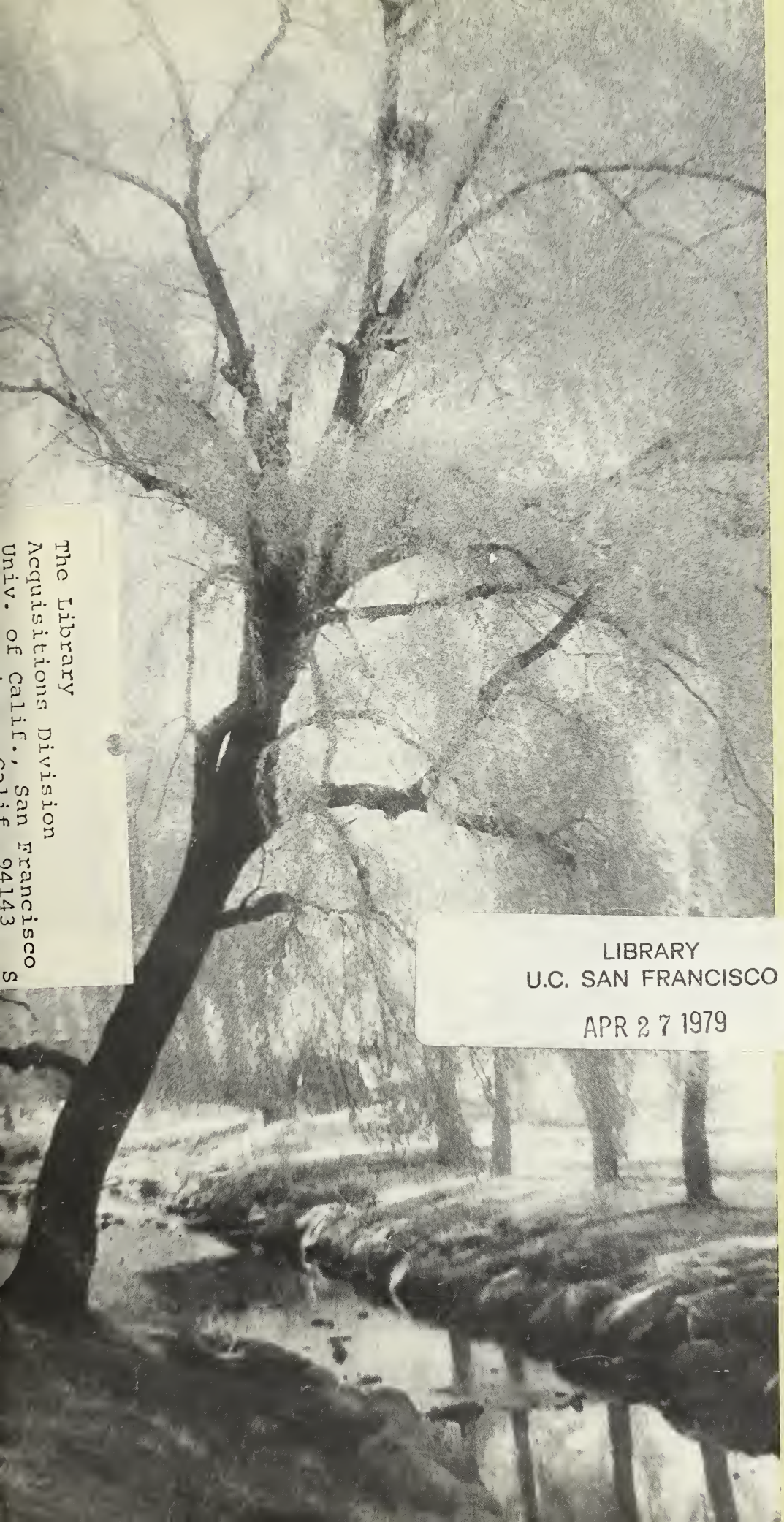
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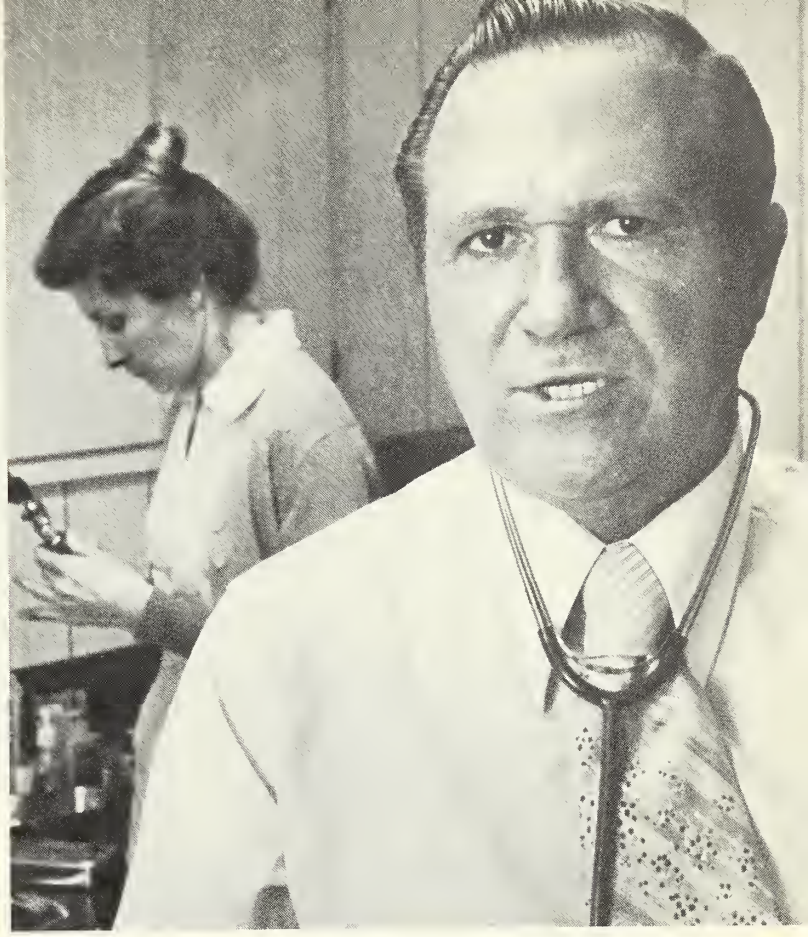
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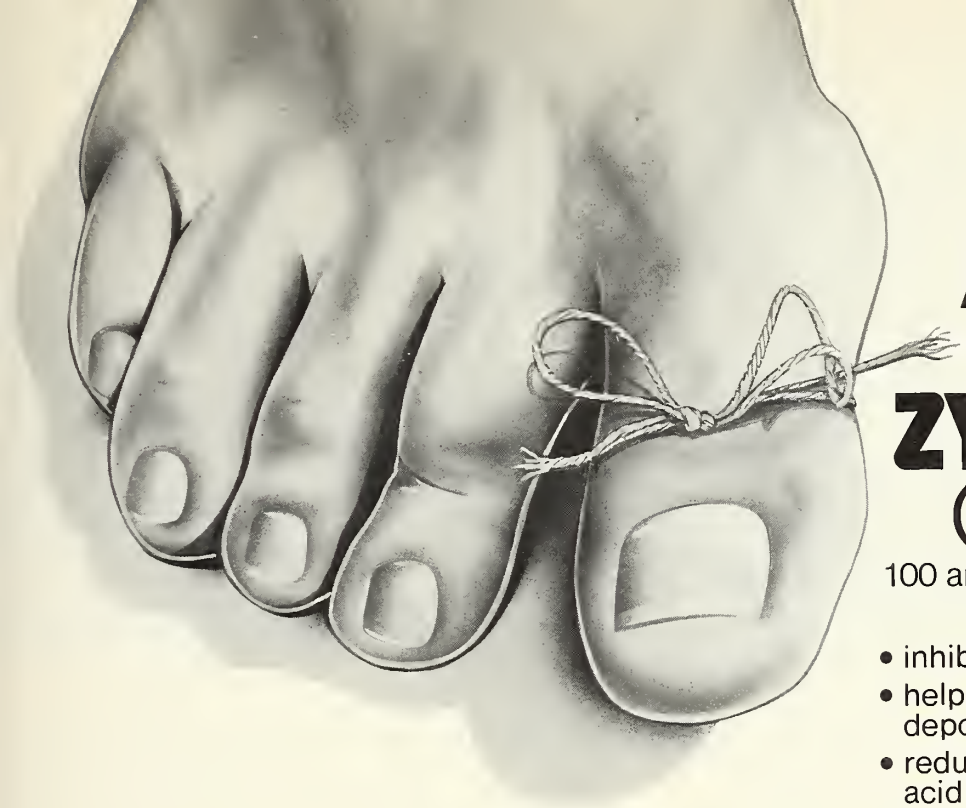
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Zyloprim® (allopurinol) is intended for:

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4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

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WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.

A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease.

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It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.
Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.
A few cases of alopecia with and without accompanying dermatitis have been reported.
In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim® (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

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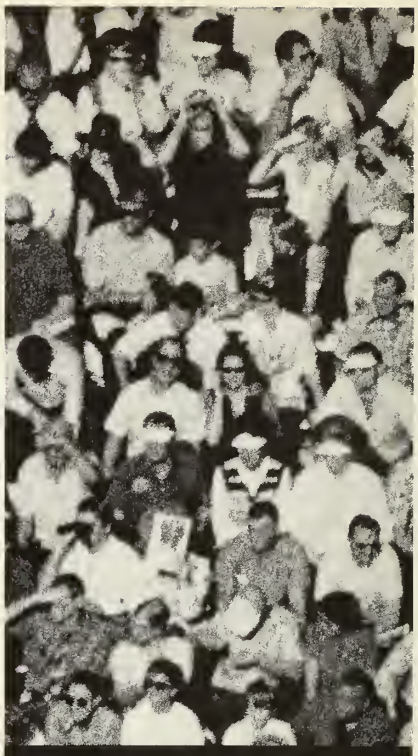
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EDITORIALS

Medical Education in South Jersey

The status of medical education in our State—particularly in southern New Jersey—may not be clear to all physicians. The College of Medicine and Dentistry of New Jersey's schools in the north (CMDNJ-New Jersey Medical School at Newark) and in the central part of the state (CMDNJ-Rutgers Medical School at Piscataway) are well known. These are four-year programs awarding an M.D. degree.

There are currently two degree-granting programs of medical education in South Jersey: the CMDNJ-New Jersey School of Osteopathic Medicine and the South Jersey Medical Education Program of the CMDNJ-Rutgers Medical School.

The osteopathic program, a four-year medical school, awards a doctor of osteopathy (D.O. degree). This is an accredited school, which was established as a unit of CMDNJ in December, 1976 by the legislature and the Governor of New Jersey with the approval of the New Jersey Board of Higher Education. The first class of D.O. candidates was admitted in 1977; there are now 52 students in this school with plans to admit 32 more in September, 1979. The first two years of training (the basic science years) currently are taught at the facilities of the CMDNJ-Rutgers Medical School, Piscataway. The third and fourth years (the clinical program) are offered at affiliated hospitals in South Jersey.

The allopathic medical education program, an extension of the CMDNJ-Rutgers Medical School, awards an M.D. degree. The first and second years are spent at the Piscataway campus and the third and fourth years at the Cooper Medical Center, Camden and other South Jersey hospitals.

As a unit, the osteopathic and allopathic medical programs in South Jersey have been referred to as the "medical school without walls." At present, the Board of Trustees of CMDNJ and the Chancellor and the New Jersey Board of Higher Education are faced with further decisions, many of which are related to "walls." In North Jersey, CMDNJ recently completed its $200 million Newark campus for teaching medicine, dentistry, and biomedical and allied health sciences. In Central Jersey, the expansion of the Middlesex General Hospital, New Brunswick, which has been identified as the Rutgers Medical School core teaching facility, will round out the current needs in that part of the state. The remaining problem is the status of the physical facilities in South Jersey.

CMDNJ's Board of Trustees, in December 1978, reaffirmed its commitment to the continuation of the M.D. teaching program at Cooper Medical Center, with further plans to expand to the level of 32 students in each of the third and fourth years of medical education. It also approved a plan to unify geographically all four years of the osteopathic medicine program on a location in South Jersey. The site, yet to be determined, will be either renovated existing buildings or a newly constructed facility, depending upon the decision of higher authority. The alternative to the acquisition of "walls," i.e., continuation of the first two years of the osteopathic medicine program in Piscataway, was rejected by the CMDNJ Trustees for a variety of reasons.

What has all of this to do with the Medical Society of New Jersey? The present position of MSNJ, as set forth by the Council on Medical Services, and approved by the Board on September 17, 1978, is as follows:*

"1. That there is need to increase the basic science capability by 100 students so as to graduate 400 new physicians in New Jersey each year.

"2. That the existing basic medical science facilities in Piscataway and Newark should be expanded.

"3. That, for the present, there should be no establishment of a basic science facility in South Jersey; if it is necessary ultimately to establish such a facility, it should accommodate both osteopathic and allopathic students."

The major opponents to the establishment of a basic science structure in that area are the Coalition of Southern New Jersey Medical Societies. Whether their feelings of opposition are emotionally based and relate to such narrow issues as hospital-practice privileges or broad issues such as allopathic versus osteopathic medical education, they are genuine and need to be considered.

At the forthcoming annual meeting in Atlantic City, these questions will come before the House of Delegates for debate. The New Jersey Board of Higher Education already has heard testimony from proponents of both sides of the subject. Undoubtedly, the Board will make its decision despite the official position of MSNJ. Be that as it may, the membership at large and the House of Delegates must be well informed on the issues. "White papers" on the subject will be available and should be studied carefully prior to May 12, 1979.

Each delegate, whether he be from South Jersey or elsewhere, should express his opinion at the time of the annual meeting. A.K.

*Trustees' Minutes. *J Med Soc NJ* 76:219, March 1979

Government Control and Public Opinion

In a 1977 survey, the opinions of physicians and the public on critical health issues were obtained and compared.* Such an evaluation of the perceptions of the provider vis-a-vis the consumer is important in order to recognize who is out of step with whom, if such a problem exists.

The major findings of the consumer survey were as follows:

1. The leading health care issue on consumers' minds is the cost of health care and cost-related issues. Two out of five Americans felt that their health care costs in the previous year were consistent with their expectations, while an equal number felt that they had spent more than they expected for health care in 1976.

2. The majority of the public is receptive to the idea of catastrophic insurance, Medicare, Medicaid, and a family premium plan of health care costs.

3. Both national health insurance and a national health service are considered "fair" or "poor" ideas by most Americans.

4. Consumers give strong support to the idea that physicians should post their basic fees in their offices, but most do not believe that government should play a role in regulation of doctors' fees.

5. Almost 90 percent of persons surveyed believe that doctors should be required to pass periodic relicensure tests.

6. The majority of consumers feel that the medical profession is both available and accessible to them for their normal health care needs.

7. The public is divided equally in opinion as to whether doctors or the federal government controls medicine.

8. Most consumers view lawyers as the major instigators of the increase in malpractice suits.

9. Approximately half of all consumers feel that their doctors are willing to spend time talking with their patients and are dedicated to their work.

10. Consumers feel that doctors should be totally frank with them both about their illnesses and the medical costs involved.

11. Forty-six percent of the consumers are willing to give the doctor the final say in treatment decisions compared to 40 percent who feel that the patient should have the final say.

On the issue of health care costs, newspaper releases from the planners at the Department of Health, Education, and Welfare in recent weeks strongly suggest that they prefer to *form* public opinion on issues rather than *conform* to public wishes—the antithesis of a democratic society. In a phased proposal which is being considered for presentation to Congress in the present session, a key premise is to mandate physician acceptance of set rates for services. Initially, the government rigidly would set and control physician payment for services to the "aged and poor," but this will be followed in short order by a regulation which will require physicians to accept "set rates for all approved private health insurance plans."

Such proposals, even though they may be patently discriminatory and contrary to the wishes of the public at large, clearly represent major ambitions of the power-hungry political forces which feel that government, not physicians, should control medicine.

The overall goals of the planners, i.e., putting a lid on health care costs, cannot be obtained without limiting greatly all services provided in or out of hospitals and by stifling medical research and the translation of new (and often costly) discoveries to patient care methodology. Government intervention in such matters historically never has contained costs. After all, the addition of catastrophic illness coverage for all employees, expanding Medicare benefits, and extending Medicaid coverage—desirable though they may be—will mushroom health care costs. All one needs to do is look at the unexpectedly high costs of the New Jersey prescription plan for the elderly on limited incomes for evidence.

Just the added bureaucracy to set up and enforce the limits on physician fees on a national basis will be no small matter to reckon with from a dollar viewpoint. This easily could wipe out any so-called savings in health care expenditures through the need for more government employees, more paper work, and more computer services.

The major stumbling blocks to control of health care costs —*government intervention* and *inflation*—seem to be low priority items in the minds of the politicians euphemistically referred to as "health planners." Instead, the public is being sold a bill of goods, which, like apple pie and ice cream, has a present cost and a future danger to the American public.

The government spokesmen like to tell their constituents that American medicine is out of touch with reality. It should be very apparent to that same public that "solutions" of this type endanger their freedom to an extent that cannot be measured in dollars.

A.K.

**Health Care Issues: Physician and Public Attitudes.* Chicago, AMA, 1978.

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Marihuana and Paranoid Disperception†

ELMAR G. LUTZ, M.D., Wayne*

Clinical experience with 60 male and female adolescent or young adult chronic marihuana smokers indicates varying degrees of cognitive disruption with paranoid development as a common sequence of chronic marihuana use. This may include severe delusional and hallucinatory behavior. Other pertinent clinical and laboratory studies, neurophysiological aspects and social implications are discussed. The author considers marihuana to be a dangerous drug and a major public health hazard.

"Marihuana is no more dangerous than alcohol or cigarettes and should be legalized for anyone over the age of sixteen," so declared anthropologist Margaret Mead in 1968. Ever since, similar statements have been made frequently and many recent articles give the public the impression that the use of marihuana (cannabis sativa) need not be or is not dangerous. This conclusion cannot be supported by my clinical or my patients' subjective experience. Chronic use of marihuana leads to many prolonged perceptual-cognitive distortions. Paranoid reactions and development of a paranoid lifestyle with potential for psychotic decompensation are common.

CLINICAL EXPERIENCE

This article was prompted by a recent new patient intake within one week of three paranoid male adolescents (aged 13, 17, and 17) and one paranoid young man (aged 22) whose schizophreniform derangement was causally connected to frequent marihuana smoking. All four had used marihuana nearly daily for two to four years with gradual personality change and paranoid development which was noticed by teachers and bewildered parents.

Two patients were overtly psychotic. One was turning around the loudspeaker in my office, looking behind pictures for his hallucinatory enemies. The day before at school he was found to be behaving in a bizarre manner and eating lawn grass. Another patient, just returning from a long "pot session" in college, wanted to have his name changed "to find himself." He presented combined paranoid and catatonic symptoms. One patient recently had dismissed his boyfriend of long standing with the remark: "I don't like you anymore, you try to analyze me." It is of clinical interest that three of these four patients had electroencephalographic abnormalities with the main finding of left midtemporal negative spike foci.

Over the past nine years, I have treated and/or examined 60 patients, 50 males and 10 females, 13 to 28 years of age, of heterogeneous background, whose psychiatric disturbances on close investigation were caused or substantially precipitated by the more or less chronic use of marihuana varying from seven months to five years. Only five of these patients had a prior history of ego-fragility, predisposition to psychosis, or family history of psychosis.

During the same time period I have examined and/or treated only 16 non-marihuana smoking patients with a paranoid thinking disorder in the same age group. Fourteen of these had a family history of mental illness. The underrepresentation of a positive family history in the marihuana smokers can be seen easily. This discrepancy would support a stronger than chance causal relationship between marihuana smoking and paranoid thinking disorder in the absence of any credible alternative etiology.

†See commentary, page 302 this issue.

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Approximately 150 other young habitual marihuana smokers with reported personality change and cognitive impairment came to my attention through indirect professional or non-professional contact. These persons were not examined by me but numerically could give further circumstantial evidence and support for the clinical observation of marihuana-induced paranoid disperception.

Psychiatric symptoms included perceptual-cognitive disturbances affecting time, space, self and others, mental disorganization with flight of ideas, incoherence, depersonalization and hallucinations, but most predominantly a distinct paranoid development. Preceding and accompanying these more acute psychiatric symptoms were varying degrees of more subtle personality changes characterized by loss of motivation with apathy and indifference, reduced goal-directed activity pattern with loss of desire to work, withdrawal from social activities, social stagnation, inability to concentrate, decrease in "emotional resonance," altered sense of identity, and academic failure. The latter often was the immediate reason for parental concern and neuropsychiatric consultation.

Hallucinations, as the ultimate expression of self-perceptual distortions, were found in all perceptual areas: visual, auditory, tactile, gustatory, olfactory, and somatic-kinesthetic. Assessment of hallucinatory behavior was most correct when the patient remained in treatment and, after improvement or cure, was able and often very eager to give a detailed account of his perceptual experiences.

The paranoid disorder of thought was the most common perceptual distortion and was present in all patients. It ranged from strong, unfounded suspicion to loss of facility of reality testing for suspicion, delusional referential ideation and, less rarely, paranoid hallucinatory behavior. The development of a projective cognitive lifestyle appeared to intensify with the frequency and duration of marihuana use.

A widening of the blood pressure amplitude often reflected the progressive paranoid development, a finding not previously reported. Increased blood pressure amplitude in my experience is also a frequent finding in non-marihuana-induced, untreated paranoid illness.

None of the patients had any explicit prior knowledge of marihuana's capacity to impair brain function, an impairment which often was reflected in chromaphilic scores on the Organic Integrity Test.

Treatment consisted of the discontinuation of marihuana, the use of small doses of trifluoperazine hydrochloride (Stelazine ®) and the temporary use of diphenylhydantoin, when EEG studies suggested a potential benefit from this drug, and supportive psychotherapy. Trifluoperazine HCl was used in a dosage of one mg twice daily to two mg three times daily. Larger doses of phenothiazines and more potent antipsychotic agents were not well tolerated and led to several acute extrapyramidal reactions, apparently not associated with the use of alcohol. It is possible that the marihuana-induced organic brain dysfunction lowers the threshold for neurotoxic extrapyramidal side-effects due to neuroleptics in a manner similar to that which I have described to occur in alcohol-induced brain dysfunction.[1,2]

Treatment, when accepted and continued, resulted in gradual disappearance of the paranoid behavior over several months to one to two years. There was an approximate direct correlation between the frequency and duration of marihuana use and the length of recovery time. The heavier and longer the patient had used marihuana, the longer it took him to return to normal after cessation of use. The return to reality often seemed to increase an awareness of their aloneness, limited belongingness, and unproductiveness, issues common during the turbulent teenage period. The doctor-patient interaction then became a most important component in the successful completion of treatment.

Since I did not know the patients prior to their drug-induced illness, it is difficult to estimate the degree of recovery or answer the question as to more permanent brain damage in young chronic users. My impression, partially based on the parents' knowledge of their children, is that subtle or not-so-subtle personality impairment persists for a considerable length of time. Resolution of the personality problems occurs gradually following cessation of cannabis use—if it does completely. It seems reasonable to assume that persons with preexisting mild or subclinical disturbances and marginal psychological adjustment may be at increased risk to develop definite neuropsychological deficits after exposure to marihuana.

DISCUSSION

Clinical and laboratory studies—Several studies corroborate my clinical experience. Ames[3] described five of ten subjects receiving marihuana in an experimental situation as experiencing delusions. Ginsberg[4] admitted that paranoia during marihuana reaction is common but stated that it is limited to concern about police raids which has a basis in reality and is thus only heightened anxiety. Keller[5] described four individuals who reported they had delusions while using marihuana. In a retrospective questionnaire study involving 181 individuals, Keeler and Moore[6] found that paranoid phenomena during marihuana use are common and are not limited to concern about a police raid. One group of subjects was comprised of university undergraduates in good standing and the other of individuals with an overt "counter-culture" appearance who were located in a "hippie" bar. They felt the use of such disparate groups avoids the possibility of basing generalizations on only one type of population. About two-thirds of the student group and ninety-five percent of the "counter-culture group" experienced suspicion. Over half of the subjects had suspected their friends were playing some trick. There were no differences in reports of paranoid items between women and men. According to Keup[7] paranoid reactions often are seen with an overdose of marihuana and as a regular part of a "bad trip" due to cannabis, especially in patients with a low threshold for potential psychotic behavior. Twelve cases of acute toxic marihuana psychosis were reported from Vietnam by Talbott and Teague.[8] Each case was characteristic of acute toxic psychosis with organic features and ten showed paranoid symptoms including suspiciousness, referentiality, and delusions or hallucinations. These clinicians advised considering cannabis derivatives as a causal or precipitating agent whenever a young person presents with an acute toxic psychosis with paranoid features.

Three groups[9] have studied the effects of oral doses of synthetic tetrahydrocannabinol (THC) on man. This synthetic substance, the active ingredient in marihuana, as well as chemical techniques for quantifying its content in marihuana have made possible pharmacological studies which provide some precision in dose. Previously divergent marihuana research findings could be explained by the built-in variability that made marihuana study more difficult than other investigations. Marihuana is a "mishmash" of more than a dozen cannabinoids, about 30 terpens, assorted sterols, and variable amounts of other compounds. Each

sample tends to differ from the others.

These laboratory studies, of relatively short duration, disclosed the following *perceptual and psychic changes:* Euphoria was most intense in the subjects of the Palo Alto Veterans Administration Study who were graduate students. It was less pronounced in the Lexington series, which used imprisoned former drug addicts, or in the NIMH Group of subjects, who were prisoners at a correctional institution. Sleepiness was a constant finding; deep sleep often followed higher doses. Hearing was less discriminant, time sense was altered, and many visual distortions appeared. Thinking and concentration difficulties, dreamlike states, and depersonalization were common. Many of these symptoms resembled those produced by psychotomimetics such as lysergic acid diethylamid (LSD), mescaline, or psilocybin. Psychometric tests showed reduced accuracy in the drawing test and a slowing of performance against time in the arithmetic test.

Clinical studies using marihuana extracts were done at the University of Utah [10,11]. These investigators, too, were impressed with the disruptive effects of marihuana on some of the most sensitive functions of the brain such as recent memory, logic, and sequential thinking. Performance was impaired in complex reaction time, digit-code memory, time estimation, hand steadiness, and reading comprehension. They have noted, as have others, a great response fluctuation during marihuana intoxication with waxing and waning attention. Impairment was explainable by loss of selective attention thus affecting the acquisition process involved in the storage of information, immediate recall, and systematic thinking.

A study of the effects of marihuana on 38 adolescents and young adults by Kolansky and Moore[12] suggests that moderate-to-heavy marihuana use may lead to "ego decompensation ranging from mild ego disturbance to psychosis." There was no predisposition to mental illness in any of these 38 patients prior to the development of psychopathological symptoms after marihuana use. The degree of severity of symptoms ranged from mild to severe, but all patients consistently demonstrated a stereotyped complex of symptoms: poor judgment, limited attention span, confusion, anxiety, depression, apathy, and, often, slow slurred speech. In all cases, symptoms began with cannabis use and disappeared within three to 24 months after cessation of drug use.

The authors concluded that the psychologic disturbances and behavioral changes were directly attributable to the habitual use of marihuana. They regard the use of marihuana by adolescents as disruptive of normal psychologic development during this critical developmental period. As a consequence, the adolescent may reach chronological adulthood without achieving adult mental functioning or emotional responsiveness. Aware of the claims that large numbers of adolescents and young adults smoke marihuana without developing symptoms, these clinicians pointed out that these claims are unsupported by scientific evidence, that no judgment can be made without definite psychiatric history-taking and examination. They believe that there is organic brain disease associated with marihuana use.

Significant impairment in performance of both mental and motor tasks was found in other studies.[13-15] Such studies showed that the marihuana smoker is as much a menace on the road as the person with acute alcohol intoxication is known to be. A recent study[16] on how smoking marihuana affects airplane pilots may be particularly interesting to those who consider the marihuana controversy closed and marihuana smoking totally safe. The authors studied the effects of marihuana intoxication on the ability of 10 certified airplane pilots to operate a flight simulator. They used a randomized double-blind crossover design to compare the effect of active versus placebo marihuana. They found that all ten pilots showed a significant decrease in measurements of flying performance 30 minutes after smoking active marihuana for ten minutes. For a group of six pilots tested sequentially for six hours, a non-significant decrease in flying performance continued for two hours after smoking the active drug. The most important factor appeared to involve marihuana's ability to affect short-term memory and sense of time. After smoking the active marihuana, the pilots often forgot where they were in a given flight sequence or had difficulty recounting how long they had been performing a given maneuver, in spite of the presence of written instructions and a stopwatch. Marihuana also appeared to cause alterations in concentration and attending behavior; the intoxicated pilots would concentrate on one variable to the exclusion of other variables or perhaps attend to intrusive thoughts. Thus, subjects exhibited a complete loss of orientation, at times, with respect to the navigational fix, resulting in grossly unpredictable flight performances. This loss of orientation occurred when the pilots were either daydreaming, "lapsing," or focusing on a certain part of their specified routine to the exclusion of other required activities. The authors conclude that the effects of marihuana on flying performance may represent a sensitive indicator of the drug's psychomotor effects. In the demanding task of flying one sees more clearly the disruptive effect of marihuana on the cognitive functions.

A report presented to the National Academy of Science and the National Research Council in 1967 described the effects of varying doses of THC consumed by smoking and concluded: "It long has been known that marihuana and hashish can cause psychotic reactions, but usually such reactions were ascribed to individual idiosyncrasies rather than being usual or common reactions to the drug. The data in these experiments, however, definitely indicate that the psychotomimetic effects of THC are dependent on dosage and that sufficiently high doses can cause psychotic reactions in almost any individual." "Psychotic reactions after smoking marihuana under the usual conditions in the United States appear to be rare, but the low incidence of such psychotic breaks may reflect nothing more than the low THC content of most of the marihauna available in the United States."

An expert scientific group of the World Health Organization recently described both objective and subjective reactions to cannabis. Subjective reports describe increased sensitivity to taste, touch, smell, and sound, while objective studies reveal some impairment of cognitive functions at high dosages. The appearance of some psychiatric symptomatology becomes apparent at high dosages among some users. The major manifestations include paranoid ideas, illusions, hallucinations, depersonalization, delusions, confusion, restlessness, and excitement.

It is of confirmative interest that the clinical syndromes described for marihuana in the laboratory correspond closely to those reported by street users.[17] Tart, using a questionnaire method, described the clinical aspects of the social use of the drug. The most frequently reported symptoms and signs were: paresthesia, floating sensations, depersonalization, weakness and relaxation, perceptual changes (visual, auditory, tactile), subjective slowing of time, flight of ideas,

difficulty in thinking and loss of attention, loss of immediate memory, silliness, euphoria, and sleepiness. Other common symptoms, which are not verifiable in the laboratory, were claims of increased perception and insight as well as increased sexual desire, performance, and enjoyment. The resemblance of many of these effects to those of other hallucinogenic drugs suggests that doses of marihuana, used socially, are fairly high and not innocuous.

Neurophysiological and neuropathological considerations—Studies conducted in lower animals (dogs, rabbits, cats, rats) indicate that brain recordings from deep nuclear masses are affected more than surface recordings by the active ingredients of marihuana.[18-20] The first study of the effects of marihuana on the function of deep brain structures of subhuman primates was done by Heath.[21] In studying the effects of marihuana on deep and surface electroencephalograms of rhesus monkeys, Heath found exposure to smoke of active marihuana "consistently induced distinct recording changes in the septal region, occasionally accompanied by changes in recordings from the cerebellum, postero-ventral lateral thalamus, hippocampus, and orbital and temporal cortices." Only generalized EEG changes, consisting of slight shifts in the dominant frequency were obtained in association with the other agents (alcohol, amphetamines, tobacco, inert marihuana) used in the study.

The scalp EEGs of the monkeys only minimally reflected the profound activity occurring at deep sites. This finding corresponds with data obtained from a study of marihuana in a severely ill psychiatric patient in whom deep and surface electrodes were implanted for diagnostic and therapeutic purposes.[22] Earlier reports also mentioned negligible scalp EEG changes in humans in association with smoking of marihuana.[23,24] None of the control substances used in Heath's[21] study induced the notable EEGs from subcortical neural sites that have been identified with emotional expression[25]. Psychotomimetic chemicals that grossly impair behavior of monkeys, resembling psychotic states of humans, have induced abnormal spiking and slow wave activity in the septal region like that recorded from the septal region of psychotic patients.[26,27] In both studies[21, 22] smoking of marihuana altered activity in the septal region, and occasionally interconnected deep sites, from which a distinct type of pathological activity, in the form of spikes and slow-waves, had been recorded consistently in a larger number of patients during periods of psychotic behavior. Heath[21] indicated that the pleasurable feelings associated with marihuana may be related to activation of the septal region and other neural sites involved in emotional expression. Evoked potential studies have demonstrated a direct functional relationship between the septal region, where activity is correlated with pleasure, and the relay nuclei for several sensory modalities.[28] This relationship between pleasure sites and sensory nuclei also provides physiologic explanation for the consistent clinical observations that severe disruptive behavior is associated with perceptual disturbances and that impaired perception can induce disruptive behavior. "When activity of the septal region is impaired, however, deleterious behavior signs and symptoms emerge in the form of reduced motivation and anhedonia, often accompanied by perceptive defect and thought disturbances characteristic of reduced awareness."[22] The impaired activity of the septal region is accompanied by epileptiform activity. This is of clinical interest since several of the chronic marihuana users I treated demonstrated primarily midtemporal spiking on the surface-EEG and probably benefited from diphenylhydantoin. It is my clinical impression that epileptiform discharges, recordable on the scalp EEG, become more frequent with prolonged marihuana use. Several investigators found that THC has anticonvulsant effects in animals.[29-32] Clinical reports, contrary to results in animals, tend to indicate that THC does not have anticonvulsant effects in human epilepsy.[33,34] It is thus conceivable that the chronic use of marihuana possibly preferentially impairs, among other subcortical structures, the septal region and with it the functional relay for sensory modalities. This could occur by direct toxic effects or by induction of immunological aberrations which also are implicated in the causation of schizophrenic reactions.[27] In a recent study the blood of 34 marihuana users showed antibodies that were present in only one of 34 non-users.[35] Heath's[21] findings of profound electrophysiological activity occurring at deep sites, reflecting extensive involvement of subcortical structures, can be related to Stevens'[36] neurophysiological model of schizophrenia. Stevens proposed a subcortical neurological focus for disordered thought in schizophrenia. She hypothesized a breakdown in neostriatal and limbic striatal "gates" ("filtering structures") interposed between the cortical mantle and afferent fiber tracts of the motor, sensory, and visceral systems. Hyperattentional states may occur in schizophrenia when the gate is "overwhelmed" and normal inhibitory abilities are diminished or lost. The patient cannot keep competing stimuli and associations out of consciousness. Coherence of thought, and thus the patient's sense of continuity and identity are disrupted. Disorders of thought appear to result from malfunction of subcortical CNS activities.

Is there anatomical structural damage to the CNS? The storage of THC and of the other natural cannabinoids and their metabolites in the brain has not yet been related widely to permanent brain damage but evidence of structural damage is emerging. However, it is difficult to show permanent brain damage histologically for any drug. Campbell *et al.*[37] reported pneumoencephalographic evidence of cerebral atrophy as shown by increased cerebral ventricle size in ten young men with histories of heavy cannabis use. Four had neurological symptoms, and five had some degree of intellectual impairment. Campbell and his associates thought that far too much attention has been paid to psychological and behavioral disturbances, without relating these to the possibility of permanent damage to the brain. For many years, they comment, it was not realized that cerebral atrophy was a hazard of professional boxing. "We suggest that a similar state of affairs is happening in relation to drug abuse." "Their patients exhibited symptoms much like those seen in encephalitis lethargica in which the basal ganglia, midbrain, thalamus, and floor of the third ventricle are usually affected." There was a 25 percent average increase in ventricle size compared to controls which might involve cellular necrosis of 15 to 20 percent of the brain cells. Heath[38] found a measurable loss of brain substance in monkeys who had been exposed to marihuana smoke, but the count of brain cell loss has not been completed yet. Anatomical structural alterations found during some postmortem examinations suggested irreversible brain changes.[39] Two recently published computerized transaxial tomography (CT) studies of marihuana smokers showed no significant brain changes that would indicate brain atrophy[40,41] but Co and associates recommend caution in interpreting or comparing their findings to those of Campbell[37] whose patients had neurological or intellectual impairment. The patients of Co and his associates did not. They mention that the cannabis may have

been of low to moderate potency and conclude "that young men with extensive exposure to cannabis over a number of years do not necessarily show evidence of cerebral atrophy." Kuehnle and associates indicate that their findings of a lack of structural damage to the CNS, as determined through the CT scan, do not rule out the possibility that marihuana may produce alterations in brain function that cannot be measured adequately with this technique. However, Jones'[38] review of one set of measurements shows that there is ventricle enlargement that clearly is related to marihuana use. No data have been made available to him from the other study. "Ventricle enlargement associated with marihuana use should be taken seriously. I think we are looking at a cause and effect relationship, but it will be years before we get in humans what Heath gets in animals."[38]

The Nobel prize winner Julius Axelrod of the National Institute of Mental Health discovered the cumulative storage of THC in fat, where, in rats, the accumulation is ten times greater than in any other tissue. The importance of fat localization in explaining the duration of action of drugs has been shown for DDT. DDT reaches maximum levels in the fatty tissue of man only after one year of ingesting the amounts usually found in food. The dangers of the accumulation of the metabolites of cannabis are obvious. Prolonged tissue storage may explain the insidious onset of behavioral changes and the prolonged perceptual and behavioral deficits after cessation of drug use and may be responsible for long-term organic brain damage.

Aspects of chronicity and social implications—In the late sixties and early seventies, we were bombarded with the conclusions of psychologists and sociologists most of whom maintained that there was nothing dangerous in smoking marihuana moderately. It widely was held that there was less cost to the body from marihuana than from alcohol. Along with this came the pseudotherapeutic offensive and rationalization, which argued that the mind reached a new serenity fueled by "pot" (marihuana), enhanced insight, and expanded self-awareness and self-expression. Smoking marihuana became an anti-establishment symbol and even fashionable chic. Compared to the more dramatic behavioral changes associated with heavy LSD use, marihuana was thought to be harmless. While sufficient data are available about the acute effects of cannabis use similar to LSD, and laboratory findings to date generally correlate well with user reports, much less is known about the implications of long-term chronic use. In few experimental studies has marihuana been administered to humans for extended periods. These periods have been limited at most to a few weeks. Also, earlier studies of both acute and chronic use provided no indication of the exact amount of psychoactive material involved, making it difficult to compare these findings with those of contemporary research. Increasingly, clinical accounts of chronic marihuana use, however, reveal disturbing findings indicating effects on the personality associated with prolonged use: apathy, loss of motivation and desire to work (the amotivational syndrome), loss of intellectual function and judgment which includes the prejudicial, or better, deluded bias against the potentially harmful effects of cannabis. The victims become goalless and slovenly, and suffer an inability to express themselves in meaningful verbal terms. Their perceptual dysfunction leads to pseudocommunication with shallow interpersonal relationships. Attentional deficits lead to non-acquisition of substantive knowledge, inability to master techniques, and impairment of higher, specifically human, goal-directed behavior ("navigational fix") compromising the ability of the young individual to make an adequate adjustment to a complex society. While it may be argued that individuals with these manifestations may have developed them with non-use of marihuana, available evidence at this time does not allow this assertion.

In one study,[42] despite nearly identical backgrounds, the heavy drug users were judged to have a much poorer work adjustment in that nearly half were either unemployed or were engaged in menial work far below their potential. Heavy users also reported inability to master new problems, considerably more difficulty relating to women, and poor to fair heterosexual adjustment. Campbell,[43] a member of the Canadian Commission of Inquiry into the Non-medical Uses of Drugs, expressed concern about the effects of marihuana use among college students.[43] In over four years of observation he found that the greater the students' use of cannabis, the greater the tendency for their grades to drop. Reviewing examination papers and essays written before and after periods of heavy marihuana use, he found a deterioration in the way the students handled material, i.e., a "lessening of the quality of abstracting and synthesizing." This effect disappeared some months after stopping heavy marihuana use.

Soueif,[44] Chairman of the Psychology Department at Cairo University (Egypt), reported results of 12 psychologic tests carried out on 850 long-term users and 839 non-users among Egyptian prison populations. On the whole, the users scored lower than the controls on most of the test variables, and this appeared to be most evident in the younger, better educated, and more urban subjects. Statistical differences in intellectual function were seen among users under 25 years of age but in few of those over 35. Impairment of cognitive skills as a result of chronic cannabis use is most readily detected during adolescence, when intellectual growth is still present. Intellectual abilities are known to peak in the early twenties and decline thereafter.

Since many of the drug users are recruited from social strata of our youth most endowed with potential intelligence and opportunity, the future loss to society, in terms of productivity and stability, of a large number of citizens has to be of considerable social consequence. Misspent youth during formative years cannot be made up. Chronic marihuana use often leads to vagabondage and chronic psychosis as is well known from experience in the Middle East, Orient, and North Africa.

Immediate social implications of cannabis use include family and other social disruption, drug-related hospitalizations, law-enforcement problems at great public expense, hazardous driving, and difficulties in handling other machinery due to disturbed depth perception and increased auditory and visual reaction time.

While this study focuses on the toxic effect on the central nervous system, demonstrating marihuana's disruptive effect on the cognitive function of man, other medical findings, possibly indicating other health hazards as a result of marihuana smoking, should be mentioned briefly. These include retention of Δ^9-tetrahydrocannabinol in tissues, particularly in fatty tissues such as brain and testicles, for as long as eight days,[45] lowered sperm counts and testosterone levels to the point of temporary infertility,[46] impairment of cell-mediated immunity,[47] reduction of DNA and RNA synthesis,[48] chromosome breakage,[47] otorhinopharyngitis, uvular edema, bronchitis, emphysema, premalignant lesions in lung tissue,[47] chronic diarrhea, abdominal cramps, weight loss,[49] and decreased myocardial oxygen delivery with decreased exercise time until onset of anginal pain.[50]

LEGAL SANCTIONS AND MEDICAL USES

In virtually all societies with extensive cannabis use, legal sanctions against both users and distributors have been developed. All the international conventions of scientific and legal experts assembled since 1924 under the aegis first of the League of Nations and then of the United Nations have recommended that cannabis derivatives, as well as opium and coca-leaf derivatives, be classified as "stupefying drugs." Such drugs are considered to be profoundly destructive to man and society. These conventions also recommended that private possession for non-medical use be considered an offense to be penalized. The Single Convention of the United Nations on Stupefying Drugs (1961) recommended that cultivation of the drug-type plant be eliminated over the next 25 years. These recommendations were taken at the initiative of the representatives from Asia and Africa, who claimed that the widespread usage of marihuana was associated with physical and mental morbidity of a large number of their people as well as with social stagnation.[51] In February 1975 the United Nations Commission on Narcotic Drugs adopted the following resolution: "In view of the numerous findings of scientific research on cannabis (marihuana) there can be no doubt as to the harmful nature of cannabis." The resolution called on more than 100 member nations to take appropriate measures against trade and traffic in all forms of cannabis. Chief Justice Tauro at the Suffolk Superior Court in Boston, after listening to twelve days of testimony by experts from all over the world, stated in 1967, "It is my opinion, based on the evidence presented at this hearing, that marihuana is a harmful and dangerous drug."[52]

Medical use of marihuana is limited and still experimental. Researchers at Harvard Medical School and Peter Bent Brigham Hospital report that tetrahydrocannabinol may be an effective medication for controlling vomiting and nausea and for stimulating appetite in cancer patients receiving chemotherapy. Other current studies are testing the effectiveness of marihuana in the treatment of glaucoma. That marihuana secures a substantial decrease in intraocular pressure was first reported in 1971.

While marihuana should be available for medical research and treatment and while decriminalization for simple possession and use of marihuana can be argued for on grounds of individual freedom and appears reasonable, deliberate legalization of marihuana would be tantamount to social sanction and therefore may not be in the public interest.

CONCLUSIONS

Marihuana is a psychoactive substance which leads to impaired brain function. While perceptual psychological changes, defective intellectual performance and decrease in psychomotor skills even occur with experimental, casual, and recreational usage, the chronic use of marihuana can lead to all the attentional disturbances and disperceptions found in schizophrenia. It frequently leads to a progressive paranoid thinking disorder which may include severe delusional and hallucinatory behavior. This disorder of thought is induced by the hallucinogen and psychotomimetic tetrahydrocannabinol. Disorders of thought can be induced by exogenous (e.g., psychotomimetic chemicals) or endogenous (schizophrenia) substances and appear to result from malfunction of subcortical central nervous system activities and therefore disruption of subcortical-cortical homeostasis. There is increasing evidence that marihuana has deleterious effects on the functional and structural integrity of the central nervous system as well as on other physiological systems. Profoundly destructive to man and society, marihuana has to be considered a dangerous drug and legalization therefore may not be in the public interest.

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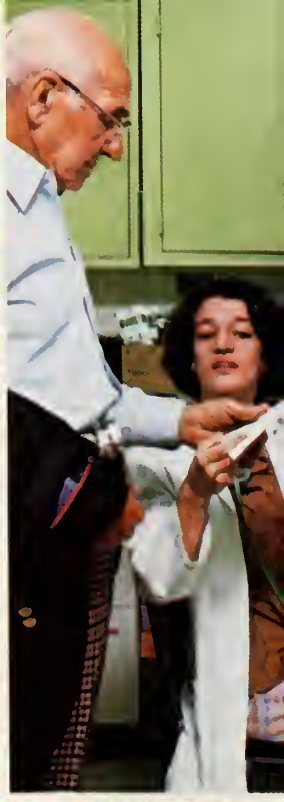
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The evidence of experience

Since October 1974 when Motrin® (ibuprofen) was introduced in the United States, it has been used by more than 6,000,000 patients with rheumatoid arthritis* or osteoarthritis. Rarely has an ethical pharmaceutical product been prescribed for so many patients in so short a time. In addition, more than 450 studies presenting new data related to Motrin have been published.

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*The safety and effectiveness of Motrin have not been established in patients with Functional Class IV rheumatoid arthritis (incapacitated, largely or wholly bedridden, or confined to wheelchair; little or no self-care).

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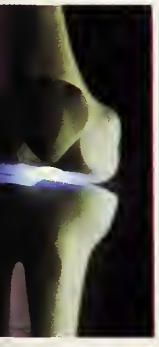












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Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin (ibuprofen) is gastrointestinal (4% to 16%). This includes nausea*, epigastric pain*, heartburn*, diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness*, headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

Incidence: Unmarked 1% to 3%; *3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

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Hemopexin in the Diagnosis of Acute Hemorrhagic Pancreatitis

SHASHI K. AGARWAL, M.D., Paramus*

The clinical suspicion of the hemorrhagic nature of pancreatitis in a patient was confirmed by finding low hemopexin levels in the blood. This is consistent with the current concepts of the role of hemopexin in the clearance of plasma heme and provides a simple laboratory method to diagnose hemorrhagic pancreatitis.

Hemorrhagic pancreatitis is a more lethal disease than edematous pancreatitis.[1-3] Accurate differentiation between the two has not been possible except by laparotomy or postmortem examination.[4] The early detection of methemalbumin in serum has been suggested as a valuable test for hemorrhagic pancreatitis.[3,6] However normal methemalbumin levels do not infallibly rule out acute hemorrhagic pancreatitis.[4] The significance of hemopexin levels in the differentiation of acute edematous pancreatitis from the hemorrhagic variety has been stressed previously,[7,8] but its mention in the American literature is rare. I report a case of acute pancreatitis in which the determination of the serum hemopexin levels lent support to the clinical suspicion of the hemorrhagic nature of the disease process.

CASE REPORT

A 39-year-old male alcoholic was admitted to the medical services of Bergen Pines County Hospital with complaints of vomiting and severe abdominal pain of two days' duration. He had been drinking about two pints of alcoholic beverages daily during the preceding few days. He was a former drug addict and was taking 60 mg of methadone daily for the past year and a half. He denied any abdominal trauma, fever, jaundice, diarrhea, hematuria, or ingestion of any other drugs. He had an attack of pancreatitis two years ago. His father had died of alcoholic cirrhosis while his mother had a history of gallstones. Other medical history was unremarkable.

On admission, he was sweating profusely and had a rectal temperature of 101.4°F. His supine blood pressure was 90/60 mm of Hg with a regular pulse of 150/minute. Respiratory rate was 20 per minute. The abdomen was diffusely tender and no Cullen's sign (discoloration of the skin about the umbilicus) or Grey Turner's sign was noted. The liver was palpable two cms. below the right costal margin; it was soft, smooth, and nontender. The bowel sounds were heard normally. Other systems were essentially normal on examination. Admission laboratory data were as follows: Hgb. 19 gms/dl, Hct. 55%, WBC 9,600/cmm, N 43, B 45, L 4, M 8; platelets "adequate"; glucose 180 mg/dl, BUN 45 mg/dl; Na 132 meq/L, K 3.7 meq/L, Cl 90 meq/L, CO_2 20 meq/L, serum creatinine 2.4 mg/dl, serum amylase 358 somogyi units, two hour urinary amylase 3620 somogyi units; amylase clearance 6.8%, serum calcium 7.5 mg/dl with an albumin of 3.5 gms/dl, serum phosphorus 3 mg/dl, serum magnesium 1.6 mg/dl, blood cholesterol 155 mg/dl, serum triglycerides 320 mg/dl. The electrocardiogram showed sinus tachycardia at a rate of 150 beats per minute with a prolonged QT interval. Chest and abdominal roentgenograms were unremarkable. Urine was negative for porphyrins.

The patient was treated with nasogastric suction, intravenous fluids and analgesics. On the third hospital day the

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laboratory parameters showed a Hgb of 13.2 gm/dl with a Hct of 38%; serum calcium of 8.3 mg/dl with an albumin of 3.6 gm/dl, serum creatinine of 0.8 with a BUN of 6 mg/dl and normal serum and urinary amylase values. Although the clinical condition of the patient had improved, a Grey Turner's sign was now noted along the left flank of the patient's abdomen. Because of the poor clinical condition of the patient, the fall in hematocrit, the initial hypocalcemia, and the subsequent development of the Grey Turner's sign, acute hemorrhagic pancreatitis was suspected. Minimal ascites was demonstrated clinically, but an abdominal paracentesis was refused by the patient. At this stage the patient's serum hemopexin was found to be 33 mg/dl (normal 50-115 mg/dl) while the serum haptoglobin was 486 mg/dl (normal 60-470 mg/dl).

Over the next week, the patient gradually improved. Repeat serum hemopexin and haptoglobin levels were reported as 80 mg/dl and 648 mg/dl respectively.

DISCUSSION

Hemorrhagic pancreatitis is characterized by the extravasation of blood into the peritoneal cavity and the interstitium of the pancreas. Hemoglobin thus released is cleaved by the pancreatic enzymes (such as elastase, lipase and trypsin) to heme and globin.[5,9] Other factors like bacteria also may play a role in this cleavage.[9] Free heme readily is oxidized to hematin, which is absorbed into the circulation and attaches to heme-binding proteins.

The presence of heme-binding proteins in the mammalian serum is well known, albumin being the first to be described.[10,11] Its interactions with heme have been well demonstrated.[12] In 1957, hemopexin, a beta-glycoprotein, was discovered,[13,14] and some recent reviews have dealt with the chemical and biological aspects of this protein.[15-17] Hemopexin is cleared and catabolized at an enhanced rate during states of plasma heme load,[18,19] and this is in keeping with the current concepts of the role of hemopexin in the clearance of plasma heme.[15,20-22] Intravascular hemolysis is the most common cause of reduced hemopexin levels in the serum.[15] However, hypohemopexinemia also is seen in porphyria cutanea tarda and erythropoietic protoporphyria,[23-25] as well as certain kidney diseases in which hemopexin passes into the urine and a few liver diseases in which the de novo synthesis of hemopexin probably is reduced.[7,26]

The reduced hemopexin levels in my patient were due to none of these causes. Hemolysis probably did not occur as the haptoglobin levels were normal. Haptoglobin is the most important protein of the extracorpuscular hemoglobin disposal system and it binds, in equimolar ratio, hemoglobin molecules released into the plasma and delivers them to the liver parenchyma.[27] Sears has shown that hemopexin drops only when the hemoglobin-binding capacity, which is dependent on haptoglobin, is exhausted.[20,28] Porphyria cutanea tarda was ruled out by the negative urine tests, while the subsequently demonstrated normal levels of hemopexin in my patient make the diagnosis of decreased production or increased excretion unlikely.

The hemopexin levels in the clinical setting of acute pancreatitis thus may suggest an hemorrhagic component, provided the serum haptoglobin levels are normal. Although serum methemalbumin levels were not measured in my patient, it is conceivable that they would have been normal. Hemopexin not only has a higher affinity for heme than albumin,[29-31] but the transfer of heme from albumin to hemopexin has been demonstrated using purified human and rabbit proteins,[32] as well as serum.[22,30,31] Hence serum methemalbumin levels may be normal in less severe cases of hemorrhagic pancreatitis, while the hemopexin levels will be low. This may explain the absent methemalbumin in the case of Winstone.[4]

Like methemalbumin, the serum hemopexin concentration also would have a prognostic value. Once hemorrhagic pancreatitis is diagnosed, the outlook is grave because of the mortality rate of 30 to 50 percent[1,2] or even higher.[3] Moreover, the continuing presence of normal levels of hemopexin is a reassuring sign because edematous pancreatitis carries a mortality of 5 to 13 percent,[33,34] and most patients will recover with supportive therapy without serious complications.

The early diagnosis of hemorrhagic pancreatitis is also significant for therapy. When surgical exploration is contemplated,[35] the accurate diagnosis of necrotizing pancreatitis is of considerable importance to the surgeon.

SUMMARY

Hemorrhagic pancreatitis is a more lethal disease than edematous pancreatitis. Accurate differentiation between the two has not been possible except by laparotomy or postmortem examination. The early detection of methemalbumin in serum has been suggested as a valuable test for hemorrhagic pancreatitis. However normal methemalbumin levels do not infallibly rule out acute hemorrhagic pancreatitis. The significance of hemopexin levels in the differentiation of acute edematous pancreatitis from the hemorrhagic variety has been stressed previously, but its mention in the American literature is rare. This report describes a patient in whom the determination of the serum hemopexin levels lend support to the clinical suspicion of the hemorrhagic nature of the disease process. The metabolism of hemopexin also is dicussed briefly. In hemorrhagic pancreatitis, the extravasated blood is acted upon by the pancreatic enzymes releasing heme and globin. Heme is oxidized to hematin and this is absorbed into the circulation where it binds with various proteins. Hemopexin is the most avid binder of hematin and undergoes increased clearance in states of increased plasma heme load. The hemopexin levels fall, and measurement of reduced hemopexin levels in the clinical setting of pancreatitis thus would suggest an hemorrhagic component. The differential diagnosis of reduced hemopexin levels in the serum also is reviewed.

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Trends in Medicine and Modern Society*

JAMES S. TODD, M.D., Ridgewood

The ultimate dilemma of American Medicine is whether to continue its independence of the past with its potential to bankrupt our health care system or to move with society toward rational solutions to common problems.

Trends in medicine and modern society is an awesome subject to address. With the current rapid changes in public attitudes, I perceive trends a good bit the way Mark Twain perceived New England weather—"if you don't like it, wait a minute." Identifying trends is difficult enough, but to predict future trends places us somewhat in the position of the famous 18th century general who said to his aide, "Quick! Go see which way my troops are going, so I may lead them!"

To a large measure the medical profession now is being hung on the pillars of its own success. The ultimate tragedy of medicine may be the profession's willingness to accept scientific change and advancement, but its inability readily to adapt to the social consequences of those changes. Three things ought to be self-evident at this point. First, we cannot do everything scientifically possible for everybody everywhere all the time; second, we or someone must decide what we are going to do for whom, and where we will do it; and third, we must all realize that personal health care is really only one determinant of health.

CHANGE IS THE DILEMMA

Parenthetically, we ought to consider ourselves lucky that the American people spend almost as much for physicians' services as they do for alcohol and tobacco. It is clear these days that the health care system has the capacity to expand infinitely, and legitimately could absorb every dollar that society is willing to make available to it. But it is also quite clear that our resources are limited and the establishment of priorities has become essential. Therefore, the issue of change in the health care system is a real dilemma. On the one hand, society has determined that health care should be a right, and it increasingly believes that medical care should be determined not by income or personal circumstances, but by some standard or need. However, we now know how to practice a style of medicine which, if extended indiscriminately, would be far more expensive than this country could or should pay and certainly more expensive than the meager improvement in health would justify.

SOME DIFFICULT QUESTIONS

Until very recently, I think that the medical profession really has not addressed this dilemma, and, in trying to do so now, it has run smack into its own ethic which says "advance the art and science and do everything you can for all who need it." Society is not blameless in this dilemma either. In their great quest for care, their pride in technological advance, and their unceasing demand for more extensive care, they too, seem to ignore the consequences of their action. With unlimited demands, and now apparently limited re-

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sources, we have some very difficult decisions to make. Is 8.6 percent of the gross national product (GNP) too much to spend for health care? Should we be doing coronary artery bypass surgery when children in inner cities are still dying of preventable diseases? How long should we preserve life for the sake of life—without cognition or quality? And who ultimately should make these decisions? These are tough questions and, so far, nobody really seems interested in asking the questions, let alone answering them. The result is a juggernaut of a health care system racing to its own destruction called bankruptcy. So far, neither the profession nor the public has perceived the need to address these problems directly, and therefore they leave a vacuum. Government exists to fill vacuums—and so enters the government. Remember that the government doesn't solve problems; it only subsidizes them.

I am reminded of what George Will, the Pulitzer Prize columnist wrote not too long ago: "the last clear success of the federal government came with the winning of World War II." The government's original response to the health situation was to subsidize; we saw more money being poured into Medicare and Medicaid. They poured in more and more money until now close to one-third of all health care expenditures come from federal funds. They supply 70 percent of all funds for research; 60 percent of the cost of physician education; 40 percent of all payments for health care services and supplies. Health is now one of the most heavily subsidized industries in this country. Add to that the massiveness of an industry that employs four and a half million people, receives 10 percent of the entire federal budget, and uses 8.6 percent of the GNP and it's not difficult to see why the interest of government is in medicine, and why that interest is going to continue.

SUBSIDY AND LEADERSHIP

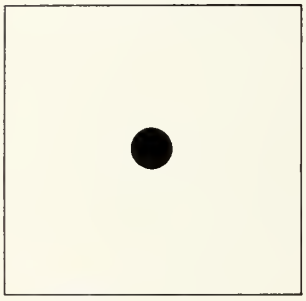
Now, the government is slowly—although some would say too rapidly—realizing that there is a limit to what subsidy can accomplish. The government is now ready to address these tough questions. But, how are they doing it? Are they doing it by assessing needs rationally and establishing priorities? Are they doing it by assuring uniform standards of quality care or by eliminating overabundant expectations? Unfortunately not. Their response is one of increasing regulation and attention to the only thing that they seem to understand—the bottom line of dollars and cents. Witness Mr. Carter's nine percent solution for hospitals; and the Office of Management and Budget's recommendation not to fund PSROs because they were not cost effective. As far as I know, they never were meant to be cost effective.

Secretary Califano describes our system as obese. Unfortunately, despite this massive federal involvement there is no evidence that it ever has been, nor is it now being, led by anyone who has any idea of the consequences of his activities. But I think that we should not be too smug about that either. Because, to a large measure, the same criticism can be leveled against the health care system. We have no visible leaders, we have no main programs, and no consensus on how to deal with these problems; indeed, a large segment of our membership denies they even exist. Do we really need a CAT scanner in every hospital? Does every cosmetic defect have to be repaired? Should the medical society assume total responsibility for the health care of this nation? Or do the citizens have some responsibility too? Somehow, somewhere along the line we have to recognize that without rational decisions from Washington, and without consistent positions from the health care system we can only expect to see more and more fundamentally important decisions made in total ignorance of their impact and their consequences. There are no easy solutions but one thing is eminently clear—that the health care system is going to change. Indeed, it is changing and this is probably the last opportunity for the profession to come together, to unite with a meaningful influence on its own destiny and for the benefit of the people they serve, which after all, is its reason for existing.

CONCLUSION

The ultimate challenge now is before us—either we move with society toward more rational solutions to common problems, or we continue the independence of the past, leading to the ultimate bankruptcy of our health care system.

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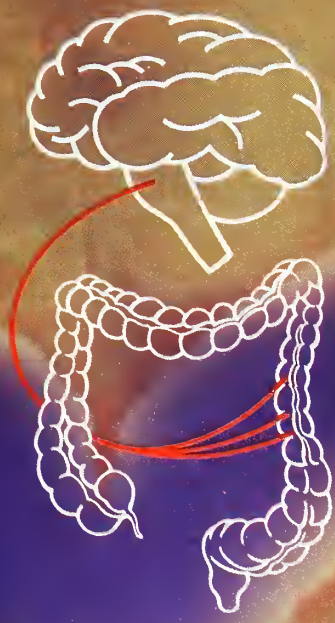
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Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially, increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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Please see brief summary of prescribing information on preceding page.

Does the American Medical Care System Need an Autopsy?*

PAUL J. HIRSCH, M.D. and
STUART A. HIRSCH, M.D., Bridgewater

If today's American Medical Care system is to survive, changes are needed, both in the mechanisms of delivery of care and in the attitudes of physicians. Physicians must recognize their obligation to provide information regarding alternatives of care to the patient and to the family; care and compassion must be demonstrated; costs must be controlled, but not to the detriment of the individual patient.

Recently there was a cartoon in a medical newspaper in which the physician is looking at the patient and saying "Not bad, not bad at all. Of course, autopsy would tell us much more." That patient might well be our medical system, and the doctor its critics. I hope that it will not be only by autopsy, that is, in retrospect, that we will determine the problems of today's American medical system. If we are to avoid this, we physicians must help, must care, must criticize, and must listen to criticism. Certainly, our system of medicine requires examination and thought.

Our system has problems. But, as with any sick patient, a great deal is right and healthy. Indeed, quality of care can be so good, that I believe that the "patient"—our American medical system—must be saved. We are using newer techniques, saving more lives, and becoming ever more skillful. Well then, what is the matter? Many reliable surveys have shown that Americans are dissatisfied with the mechanism of delivery of our medical care today; we cannot ignore this. They are dissatisfied with our *attitudes;* I fear that they are right. Indeed, this is one area where self-help, a change in the "lifestyle" of our system, could add immeasurably to its vitality.

Many of our patients are reluctant to enter the system of medical care for their problems. This is not unrealistic. When they do, they enter a system which they do not understand, in which they lose control of their own immediate destinies, in which *care and compassion* seem to be virtually lacking. Indeed, as in so much of today's life, entering the medical care system becomes a hassle, and involves fighting the system and its representatives at many levels.

The medical community can change this. The medical schools *must* change this. With all of their talk about "the total patient," the professors do not emphasize this by example, but only by lip service. I see this daily. Our young physicians are not taught to understand the apprehensions and fears of the sick patient, and ways in which their anxiety may be relieved. Obtaining a test is important, administering treatment is important, but taking a moment to explain it to the patient and his family often has no place. Indeed, a few questions by the family may label them as troublemakers. In a recent article in the *Saturday Review,* Norman Cousins asked, "Is the obligation of the doctor confined only to the patient? What about the legitimate emotional needs of those very close to the patient?" Only when those practicing and teaching demonstrate real compassion for the sick patient and for the family (for their needs are enmeshed) will the students and the house officers and the nursing staffs learn this. We must not fail in making this a part of medical care.

More than fifty years ago, Dr. Edward Trudeau stated that

*Presented before the joint session of the Sections on Orthopaedic Surgery and Family Practice, the Medical Society of New Jersey, 212th Annual Meeting, May 8, 1978. The Doctors Hirsch are clinical assistant professors of surgery (orthopedics), CMDNJ-Rutgers Medical School. Correspondence may be addressed to the authors at 720 U.S. Highway 202-206, Bridgewater 08807.

the physician's role is "to *cure* sometimes, to *relieve* often, to *comfort* always." Many physicians today are adept at *cure*, when it is possible, but lose interest when they can only *relieve* and rarely consider *comfort* their job.

In addition to compassion, the patient is entitled to *information*, and we must take the time to provide it. If there are alternatives of treatment, the patient—and his family—should know this, and the advantages and disadvantages of each. The patient is entitled to participate in decision-making which affects him.

Cost containment is a problem that I have a great deal of ambivalence about. Certainly, it is necessary. And yet, our utilization committees and other reviewing organizations are beginning to equate cost-effective medicine with quality medicine; this is not always so, and the medical profession must fight hard to maintain the distinction. I believe that the physician caring for an individual cannot limit that patient's care by considering the economic needs of society. No patient would agree to this for himself; we cannot "agree" on his behalf. Perhaps these decisions need to be made by society, through its governmental representatives; but as individuals, we must do our best. Furthermore, the concept of cost effectiveness must not be used to prevent progress in the field of medicine.

On the reverse side of this coin, however, is the need for us to be conscious of increasing costs and to avoid duplication of tests, prolonged hospitalization, and other forms of unnecessary spending. Increasing costs are providing the most potent magnet for governmental involvement in medicine. Again, an example must be set for the house officers and the students. Clinical judgment should not be replaced by screening by laboratory tests.

These then, are some of the areas in which physicians can modify important shortcomings in the pattern of medical care today. Primarily they require changes in attitude and are basic and essential, but they are not enough. We must be responsive to the needs of the society we serve.

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Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in postsympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

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Organizing To Contain Health Care Costs: The Minnesota Medical Perspective

RICHARD L. REECE, M.D., St. Paul, Minnesota

Minnesota is a state where health professionals believe in organizing to contain health care costs. This attitude is reflected in: 1) an experimental and innovative series of mergers among hospitals; 2) a tendency among physicians to form groups; 3) a willingness to accept HMOs as a competitive force; 4) an emphasis on primary care; 5) a tradition of formally organizing at all levels—hospital trustees, hospital administrators, and medical societies—to contain health costs.

Alfred North Whitehead, a Harvard professor fifty years ago, observed: "It requires a very unusual mind to undertake the analysis of the obvious."[1] At the risk of being considered "unusual," I shall begin by stating the obvious: Minnesota, where I live, is not New Jersey. The two states differ demographically, geographically, economically, and pathologically, i.e., in diseases citizens die of.

But we vary little in money we spend for health care. In 1974, Minnesota and New Jersey spent about eight percent more per capita for Medicare patients than the United States as a whole.

As states, then, we share the problem of keeping health costs in line. Today, I shall speak of Minnesota's perspective in containing health care costs. First, a few words about Minnesota.

THE STATE THAT WORKS

Minnesotans believe in cooperative enterprise between public and private sectors, in organizing to satisfy the public, and in placing pragmatism above idealogy. What works interests us, not what your politics are. Perhaps this cooperative pragmatism is why liberal Minnesota politicians and conservative professionals and businessmen generally manage to find common ground.

DISTINCTIVE FEATURES

Minnesota's ethnic heritage, its geographic isolation, its confidence in its ability to manage its own affairs, and its trust in the political process make it distinctive.

This trust is possible and even justified because Minnesotans are extraordinarily active in local and national politics. Political scientists have long noted how Minnesota citizens crowd local political caucuses and how independent Minnesota voters are. Even political party names, Democratic Farmer Labor and Independent Republicans, reflect this cooperative and independent spirit. Nationally, Minnesota voters routinely lead the nation in percent voting in Presidential elections. Because of political training and activism, Minnesota's political power exceeds the two percent of US population Minnesota people represent.

Three years ago, a *Wall Street Journal* lead article proclaimed Minnesota the "Candidate Factory"—for good reason. At the time, four of five candidates for the Presidency were from Minnesota—Humphrey and Mondale for the Democrats, Eugene McCarthy for the Independents, and Gus Hall for the Communists. And, should any of them fail, Harold Stassen was waiting in the wings for the Republicans.

HEALTH, EDUCATION, AND COMMERCE

Political activism spills over into the business and medical community. Minnesota business and physician leaders are movers, shakers, and organizers. They have good people to

* Based on a presentation before the joint session of the Sections on Orthopaedic Surgery and Family Practice, 212th Annual Meeting, the Medical Society of New Jersey, May 8, 1978, Atlantic City. Dr. Reece is Editor-in-Chief of *Minnesota Medicine* where this article was published previously—July 1978, pp. 409-415.

Table 1
Tale of Two States as Told by Basic Data

	New Jersey	*Minnesota*
Papulation	7.2 million	3.8 million
Land Area	7,800 square miles	84,000 square miles
Population Density	920/square miles	45/square miles
Ethnic makeup	Italian Americans are largest ethnic group; 11% of people are black	One of two citizens of German or Scandinavian origin; 1% of citizens are black
Climate	Temperate: average temperature, 50°F in winter, 70°F in summer	Extreme: average temperature, 10°F in winter, 82°F in summer
Economy	1 of 3 in manufacturing	1 of 4 in manufacturing
Relative Death rates/100,000 compared to United States		
All causes	100	86
Malignancies	110	91
Heart Diseases	113	86
Cerebrovascular Disease	87	95
Motor Vehicle Accidents	68	101
Life Expectancy	71 years	73 years

work with. Our citizens have a strong work ethic, a sense of self-responsibility, dedication to health and education, and civic pride.

Permit me to give you a litany of statistics to show this pride has sound basis:

- Minnesota ranks seventh in the country as headquarters for national corporations. In the Twin Cities, many of these corporations, as a voluntary civic duty, give five percent of pre-tax income to cultural institutions.
- Minnesota's highest paid public official is the President of the University of Minnesota, which is the fourth biggest university in the country.
- Minnesota secondary school students have the lowest dropout rate in the United States, and its graduates often score highest in the country in the US Armed Forces entry tests.
- Minnesota has three medical schools turning out 316 graduates each year in a state of four million. That's eight graduates for every 100,000 students. Compare this to New Jersey, which turns out 140 graduates for seven million citizens, or under two new graduates per 100,000.
- In the University of Minnesota Medical School—which has the largest organ transplant program in the world, Hennepin County Medical Center—which runs a large teaching program and one of the country's biggest renal dialysis programs, and the Mayo Clinic—one of the finest institutions of its kind, Minnesota has three outstanding medical centers. Because these institutions attract many out-of-state patients and train many physicians, Minnesota exports doctors and medical care.
- Minnesotans are notably healthy, ranking second in longevity among the other states, third in infant mortality, second in death rates, and eighth in number of deaths from heart disease.[2]

WHY THE BUILD-UP?

Why have I given you this elaborate build-up about Minnesota before even getting to health care cost-containment? Well, this is a sprawling and diverse country. To understand a state's health services you must understand its people. Health services reflect attitudes, culture, and customs of people interacting with the medical profession—which is in itself shaped by the same traditions.

Of Minnesotans' traditions and talents, perhaps the most notable is a gift for organizing public and private forces into workable alliances. Perhaps this gift is a primordial urge to huddle together against the cold. Maybe it's a herd instinct from living on thinly populated prairies. Maybe it's a community tradition from organizing farm cooperatives in a rural state. Or maybe it's because German and Scandinavian blood clumps in our veins.

For whatever reason, Minnesota health professionals are persuaded the best way to preserve the private practice of Medicine is to organize our medical societies, our relations with the rest of society, and our system for delivering care so we can compete in a modern society of interacting pluralistic organizations.

BACKGROUND SETTING

Before I describe what organizational steps we have taken in Minnesota, you may wish to review four social changes that have occurred in the United States since World War II. These changes, to my mind, make our organizational efforts necessary, relevant, and promising.

1. We have become a society of organizations—a society that entrusts every major social task (education, defense, production and distribution of goods, government services, and health care) to large organizations. Managers—"executives," "administrators," or "bureaucrats"—run these organizations and society. The most rapidly expanding organizations are in the public sector, which now consumes nearly 40 percent of GNP and employs one of five workers.

2. Because of this organizational and managerial growth, a "new class" has risen. This "new class," trained mostly since World War II, is college educated: editors, journalists, scientists, teachers, educational administrators, television commentators, city planners, psychologists, managers in the upper reaches of government bureaucracy, and public health physicians or lawyers in the public sector.

This new class shares certain characteristics: (a) they are interested in "being" someone rather than "having" possessions; (b) they are "idealistic," i.e., they do not care about money but being the elite that exercises power; (c) they feel compelled to provide common people with what they "need," e.g., mass transit, rather than what they "want," e.g., cars; (d) they have little or no experience with the market system; indeed they are suspicious of anyone, e.g., businessmen or fee-for-service physicians who deal in market transactions; and (e) they believe ultimate power should reside in the government, where they will have the major say in intervening in the private sector and redistributing power for the "common good" and "social justice." According to

Irving Kristol, a neo-conservative, the "new class": ". . .*are* the media—just as they *are* our educational system, our public and welfare system, and much else."[3] *They* also *are* competing with businessmen, professional people, tax reform groups, and populist politicians for power. Unfortunately, the "new class" has little economic common sense, i.e., appreciation of where the money comes from.

3. Power is shifting from major political parties to issue-oriented groups. This new political reality has occurred for various reasons—new ethical requirements for legislators, activities of Common Cause and consumer groups, retirement of powerful politicians of old school, and, emergence of complex technological issues such as energy, pollution control, and health care that cut across party lines and require special knowledge. *Fortune* says: "Congress has splintered today into a pluralistic collection of independents and mavericks, knowing no party loyalty."[4] This same splintering has occurred in state legislative bodies. Result? Fierce lobbying on specific issues at all levels of government. Lobbies with the best staff work, the most facts, and the best knowledge of the legislator's constituency win the day.

4. The most recent social phenomenon is the new conservatism, now manifesting itself as the "tax revolt." Because of government's poor performance in Great Society and welfare programs, disillusionments of Watergate, and inflation with increased taxation, the middle class has come to realize the government is inherently inefficient and that the burden for any system of "transfer-payments"—Social Security, Medicare, and Welfare—falls mainly on the middle class. There are never, it seems, enough rich people to go around. The middle class also feels wealth is "transferred" to the not-so-poor, who have more social benefits than the paying middle class.

RESPONSE TO SOCIAL FORCES

These, then, are major social forces organized Medicine must consider. To compete effectively politically, we must build strong organizations, hire or develop effective managers, understand how the new class thinks, concentrate on lobbying, and capitalize on the effects of the new conservatism on the public. The new conservatism has achieved intellectual respectability. That, in itself, is a breakthrough.

But to sustain this respectability, we must have reasonable goals. In health care, these would include a restructured, flexible health system that would: (1) provide reasonable choices of types of care; (2) provide financial protection and access to these choices; (3) distribute care equitably; and (4) give good care at a price society can afford.

STRENGTHS OF MINNESOTA HEALTH SYSTEM

Finally, we must work from the strengths of our particular region's health system. To do that, we must recognize those strengths. In Minnesota, these strengths are:

1. A highly experimental and innovative attitude among health care professionals—from politicians, to administrators, to physicians.

2. A well-established tendency among physicians to join groups, as noted in Table 3.

3. A willingness, both among citizens and physicians, to accept HMOs as a competitive force in the community. Minnesota has eight HMOs, more per capita than any other state. In the Twin Cities, HMOs have 190,000 members—10 percent of the population. One HMO, the Physicians' Health Plan, has 90 percent of the physicians in the Metropolitan Region participating and 23,000 members. The Health Plan broke even after two and a half years, but only after going deeply in the red the first year, then imposing these rules, some of which have been relaxed since the Plan became solvent:

Controls Imposed by Physicians on Themselves in Physicians' Health Plan

1. Paid themselves only 70 percent of standard fees
2. Asked physicians to file monthly reports detailing what they did
3. Required permission to admit nonemergency patients to hospital
4. Allowed plan to review status of hospital patients
5. Allowed plan to analyze their office practices

Another HMO in Minneapolis is a multispecialty clinic (The St. Louis Park Clinic) that employs 100 physicians and also practices fee-for-service medicine alongside their prepaid plan for 40,000 members. Physicians find these two systems of practice can be carried out side by side.

In matched populations, HMOs reduce hospitalization rates by five to 40 percent. Yet HMOs do not "maintain health" or prevent disease. As elsewhere, we have mixed emotions about HMOs. We invite them to compete, to vie for beds, and to form. But we know HMOs probably will care for no more than 20 percent of the population (if the Permanente experience is any indication). Also, for sound financial reasons, HMOs are reluctant to enroll the poor, the infirm, the old, the sick, the psychotic, or the alcoholic. HMOs have other limitations: large capital investments and years to reach a break-even point, lack of attractiveness to many patients and physicians, and a failure to catch on in rural regions. But HMOs are a legitimate alternative for care, and they do make us all aware how to cut costs, even fee-for-service physicians practicing in the same area. In Minnesota, the medical profession accepts HMOs as a competitive force, which is why, of course, Twin Cities physicians formed their own HMO.

4. More emphasis on primary care. Fifty percent of Minnesota physicians are in primary care, compared to 40 percent for the rest of the U.S. Also 65 percent of University of Minnesota Medical graduates now go into primary care. The University ranks second in the country in this regard. Finally, 42 percent of their graduates are going into family

Table 2

Money Spent per Medicare Patient. Source: Unpublished Data from Social Security Administration

Per Capita Reimbursement by Medicare (in dollars)

	All Persons	*Persons over 65*
United States	$471	$467
New Jersey	508	497
Minnesota	510	498

Table 3

	Minnesota	*United States*
Percent of Physicians in any group practice	43.7%	17.6%
Percent of Physicians in single specialty groups	13.4	6.2
Percent of Physicians in family practice groups	5.1	1.0
Percent of Physicians in multi-specialty groups	25.3	10.4

practice, versus 13 percent for the rest of the country. Each year forty of these family practice students participate in a rural preceptorship program, and 70 percent of these students return to the site of their preceptorship to practice Medicine.

5. An enlightened group of hospital administrators who are willing to change to adapt to the times. Within the Twin Cities, at least eight new organizations have formed in the last ten years for purposes of merging, affiliating, or cooperating with other institutions. Six of these organizations have budgets over $35 million and are sharing, centralizing, decentralizing, or otherwise doing what it takes to survive. They are also innovating. Minnesota is a national leader in the concept of organizing multihospital units in one system, in setting up one-day surgical units, in devoting beds to alcoholic rehabilitation (we now have more than 15 percent of all alcoholic rehabilitation beds in the country, which may be a good thing, because Minnesotans, on the average, drink 50 percent more than the average U.S. citizen), and have been leaders in setting up a National Health Rehabilitation Center, with seed money from the Norwegian government, called Vinland Center, to teach and promote positive approach toward health habits.

6. Different patterns of health facilities usage. For example, because of the flurry of mergers and consolidations, Minnesota now has fewer hospitals than five years ago, a lower occupancy rate that is dropping faster than the rest of the country, and a much lower rate of hospital outpatient visits than the rest of the country. Our hospital expenses are less, and we have fewer employees. In nursing homes, we have twice as many beds per capita, but less employees. Our average hospital cost inflation rate has been 12 percent these last five years, compared to 15 percent for the rest of the U.S.

7. A tradition of forming organizations to monitor and evaluate health care. In 1969 the Hennepin County (Minnesota's most populous county) Medical Society formed a foundation, later called The Foundation for Health Care Evaluation. The Foundation encourages physicians to participate and encourages insurance people, industry representatives, consumers, and providers to cooperate. Membership includes 2,042 physicians (nearly 1/2 the physicians in Minnesota) from the seven-county metropolitan region. At first, the Foundation dealt mostly with evaluating whether fees were appropriate. In 1972 it undertook the duty of hospital utilization review, then in 1974 was recognized as the PSRO for the Twin Cities. In the last six years, the Foundation has reviewed 6000 contested fees and reduced 63 percent of them by $332,621. It has also initiated a data system, evaluation activities, and made audit studies of such procedures as D and Cs and hysterectomies.

MINNESOTA MEDICAL ASSOCIATION AND ESTABLISHMENT OF DIVISION OF SOCIO-ECONOMIC AFFAIRS

So much for background, now a few comments on the role of the Minnesota Medical Association.

In January 1975, Congress passed Public Law 93-641, the law that divided the country into 205 Health Service Areas, each with its own Health Service Agency, to be dominated by consumers. The aim was to "democratize" health planning, to have the states set up control agencies to coordinate HSAs, and, presumably, to establish the administrative framework for a future national health insurance plan. The Association responded to this ambitious federal plan, to state legislative activity, to the malpractice problem, and other socio-economic problems, by increasing dues by $100 in 1975, $50 of which was earmarked for the new Division of Socio-Economic Affairs. The budget of this Division was $285,000 in 1976, $299,000 in 1977, and $394,000 in 1978. The 1978 portion represents about 40 percent of the Association's total budget.

In other words, the Association has devoted time, money, and resources to socio-economic affairs. Why? To meet the social changes I have described and also to cope with federal plans to reform the health care system. The Minnesota Medical Association feels, although the government is pervasive, it is not necessarily effective. Indeed, the government is surprisingly weak, especially in its knowledge of health care matters. Therefore, the government will benefit from concentrated physician knowledge, participation, and decision-making.

To form its Socio-Economic Division, the Association hired a Director, his assistant, and a staff counsel. The Division now has a staff of 11 and is comprised of three units. (See below.)

Units of Socio-Economic Division	Roles and Duties of Unit
Department of Legislative Affairs	Monitors state legislatian, represents the Association before the State Legislature, prepares analyses and summaries for Committee on Legislature, helps in deciding policy decisions, and works with staff counsel in legal analysis of bills before legislature. Usually closely follows about 40 pieces of health-related legislature through each session. This department also tracks national legislation, maintains liaison with AMA, and provides infarmation to Political Action Cammittee on vating records of Minnesota Legislative members.
Department of Health Planning	Staffs Committee of Health Planning and decides Association's approach to health planning in seven HSAs in Minnesota. Each HSA has a Board with Task Forces. Health Planning Department forms boards in parallel with HSA's task farces and appoints Reference Groups in parallel with Task Forces.[6,7] Idea is get physicians involved and ta focus their wisdom, knowledge, and advice on HSA Task Forces. By being cooperative, honest, and informative, physicians have earned respect af HSAs.
Department of Medical Services & Research	Staffs Committee on Medical Service, which deals with state and federal regulatary agencies, medical insurance carriers, HMO's, nursing home regulations, PSROs, health screening programs, secand apinion surgery projects, and cost containment. Maintains a close relationship with State Department of Public Welfare, which usually does not make majar decisions without consulting with Cammittee.

COST COMMISSION

A year ago, as an outgrowth of its Division of Socio-Economic Affairs, the Minnesota Medical Association formed a Cost Commission that included representatives of the insurance industry, business, labor, government, think tanks, institutions, and hospital groups. The Commission will deliberate for 18 months, will invite speakers and papers representing different sectors, and will publish a report of the Commission's conclusions.

I cannot say now, when we are only 12 out of 18 months into the deliberations, what the Commission will conclude. As Editor of *Minnesota Medicine,* I am not directly involved in the Commission's work. But I am indirectly involved. Our journal regularly publishes formal presentations before the Commission. Three articles have appeared—one from a University of Minnesota health economist who analyzed the alternatives we have, one from a State legislator who spoke for consumers who were worried about uneven access and excess costs, and one from an Interstudy health economist who came out strongly for restructuring the private system, i.e., offering the public its choice of competing health plans, and minimizing government regulations.

The Commission also commissioned a health economist to study Minnesota's unique health system and how it differs from other states. This 108-page report is now available and is called "Containment of Health Care Costs—The Case of Minnesota."

The idea of the Commission, according to Chairman Richard J. Frey, is simple: "We've got to get everybody involved to make changes—doctors, institutions, third parties, and patients." Doctor Frey has set up four task forces—providers, consumers, third party, and institutions—and their aim is to make specific recommendations for change. To give an example, one task force is concentrating on the pre-admission testing and in-office surgery as a means of saving money. Another has enlisted the aid of Minnesota state specialty societies. Each specialty is preparing a list of the ten most common reasons for hospitalization, the ten most common operations, and recommending how they can cut the cost of each.

OPTIMISTIC CONCLUSION

I would like to end on an optimistic note. I think it is generally agreed, even by liberals, that a comprehensive centralized National Health Insurance plan, administered in Washington, is a dead concept. A limited catastrophic insurance is much more likely. Also the rapid growth of federal bureaucracy is coming to an end. Finally there is a growing consensus that government cannot solve all problems, and among things the government does badly is regulating health costs.[12]

We are going to have to solve cost problems as individuals and as members of cooperating and competing pluralistic organizations. Leaders from different private sector organizations of Medicine are beginning to meet and talk, coordinate, and unify their approach and relationship with the government.[13] In Minnesota, we firmly believe private initiatives by physicians—through individual patient decisions, through thoughtful decision making in health care, through organizing new alternatives to deliver care, and through monitoring our own behavior—offer a better and more effective choice than government regulation.

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The Health Care System in Canada: It Works

JOHN E.BROCKELBANK, Saskatchewan, Canada

The Speaker of the Legislative Assembly of Saskatchewan, Canada describes the historical development of the hospital and medical care insurance programs of our northern neighbor. He discusses their problems and potential solutions. Life study change and prevention now are being promoted.

In this year of Our Lord 1978 I have traveled 1978 miles to address you today. This state and my province (Saskatchewan) are a long way apart and present sharp contrasts when compared to each other. Your population is well over seven million—our population is less than one million. Even more of a contrast is that our population is spread over a quarter of a million square miles, while you must be a little bit crowded with only 7836 square miles—one-thirty-second of the area of Saskatchewan. I mention these brief statistics because they are relative, in part, to the subject we are discussing.

I am honored to be invited to address this meeting of the Medical Society of New Jersey. The theme for this meeting is "Trends in Medicine and Modern Society." I am particularly proud to be here as a representative of a province which has a history of trend-setting in the health care field. I have been asked to speak about the Canadian health care system and to discuss the advantages and disadvantages of the system as well as the problems we currently are experiencing. The Canadian health care system has evolved over the course of several decades to provide comprehensive, prepaid coverage for medical care, in-patient hospital treatment, and a wide range of out-patient and extended-care services to over 99 percent of the population.

DEVELOPMENT OF HOSPITAL CARE

Before I briefly outline the historical development of our health care system I would like to elaborate slightly on the social and political context in which our health care services developed. In the first place, health care programs in Canada were established in response to a compelling need for such service. The initial impetus was provided by serious deprivation and/or unavailability of services. In most cases, government involvement in this field was in direct response to public demands for services. Secondly, the Canadian health care system is an integral part of a broader social and economic system. Government involvement in health care is not unique, but is paralleled in other fields—education, the provision of social services, welfare, pensions, workman's compensation, and others. Therefore, the health care program should be viewed within the context of the social and economic system, which virtually has eliminated any real destitution and insures at least a minimum standard of living. Thirdly, in Saskatchewan we have benefited from a collectivist spirit which was fostered by the juxtaposition of a small population on a very large territory. In order to provide services under such circumstances people had to work cooperatively. As a result of this tradition, particularly in the health care field, health service has been a very emotional issue in our province. People have not just tolerated public involvement in this area, but actually have encouraged increased government expenditure for health programs. Governments have been expected to initiate, innovate, and

*Presented before the joint session of the Sections on Orthopedic Surgery and Family Practice, Annual Meeting, the Medical Society of New Jersey, May 8, 1978, Atlantic City. Mr. Brockelbank is speaker of the Legislative Assembly in Saskatchewan, Canada.

expand their health care services. Finally, in Canada, legislators have been expected to take the lead in developing and providing services. This is a very different political climate from that in the United States where the emphasis has been on as little government as possible, and where government is expected to fill the gaps left by the private sector rather than develop comprehensive government-sponsored programs.

It is within this context that you should view the development of the Canadian health system. While some form of health insurance was available in Canada as early as 1655†, health insurance did not become a popular concept until the 1900s. In 1916, the province of Saskatchewan took one of the first steps to break down the tradition of municipal responsibility for hospital care by passing the Union Hospital Act. This Legislation permitted municipalities to combine into hospital districts for the purpose of building and maintaining hospitals. In 1917, municipalities were granted taxation powers to finance hospital care for their residents. By the mid-1940s there were more than 100 local hospital plans in Saskatchewan. In 1945 the federal government developed a health insurance proposal which would have provided financial assistance to provincial governments for the establishment of comprehensive health insurance programs. The health insurance proposal was only one aspect of a broader proposal which was not accepted by the provinces.

Meanwhile, the province of Saskatchewan decided to proceed independently and, in 1946, passed the Saskatchewan Hospitalization Act, which, when implemented the following year, constituted the first government-sponsored, province-wide, compulsory program providing hospital care in Canada. It was not until 1957, eleven years later, that the federal government reentered the hospital insurance field with the passage of the Hospital Insurance and Diagnostic Services Act. This legislation provided federal support for specific hospital and diagnostic services including acute care and services in chronic and convalescent hospitals. Initially, five provinces including Saskatchewan signed agreements under this legislation; the remaining five provinces joined in later. The hospital insurance programs provided coverage on a prepaid basis to everyone except those persons already covered by existing legislation, such as the Workmen's Compensation Act. Uniform coverage became guaranteed throughout the country, while individual provinces retained the responsibility for administering and financing the provincial share of the program's costs.

In 1966 the Canada Assistance Plan was introduced and extended coverage to certain health services such as special-care homes for needy persons. The Health Insurance Supplement Fund pays claims for health services for Canadian residents who are for some reason not eligible for provincial hospital insurance coverage. So what we have today is a hospital insurance program which provides automatic coverage on an equal basis to all provincial residents. Coverage is also portable both within Canada and elsewhere in the world during a temporary absence. Benefits extend virtually to the entire Canadian population.

MEDICAL CARE INSURANCE PROGRAMS

The development of medical care insurance programs followed a similar course. Once again, Saskatchewan was the pioneer in this field. In 1914 the rural municipality of Sarnia provided a physician with a retainer to practice in that area. The Rural Municipalities Act, which was passed in 1916, gave rural municipalities the authority to levy property taxes for the purpose of paying a retaining fee to doctors providing general practice or public health services. By 1946 there were approximately 100 such municipal physician arrangements in Saskatchewan. In the late 1930s physician-sponsored medical plans began to make their appearance. Generally, these plans provided prepaid coverage, requiring no payment by the patient at the time the service was provided.

The first comprehensive government-sponsored Medicare plan was introduced in the Swift Current region (in the southwest part of the province) in Saskatchewan in 1946. This program provided a full range of medical care services to 54,000 regional residents and included a dental care program for children under 16. These services were financed by compulsory personal and property taxes and by provincial grants. Gradually, the provincial government provided medical care services for welfare recipients and indigents. However, by 1948 government-sponsored medical care to groups other than welfare recipients was limited to the Swift Current Health region and to children in Newfoundland.

The introduction of the Saskatchewan Medical Care Insurance Plan in 1962, which marked the entry of provincial governments in this field in a major way, met with bitter opposition from the medical profession. An agreement between the government and the physicians was reached only after the physicians had withdrawn their service for a period of time. The Saskatchewan program was the first compulsory government-sponsored medical insurance program. It was based on the following principles: First, prepayment; second, universal coverage; third, high quality of services; fourth, public administration; fifth, acceptability to both the providers and the consumers of services. All essential physician services, including the diagnostic services, were covered; over 93 percent of the Saskatchewan population was protected.

While several other provinces introduced medical care programs, universal coverage was limited to Saskatchewan until the passage of the Medical Care Act, which was federal legislation. This legislation, implemented in 1968, provided for federal government cost sharing of medical services.

To qualify for cost sharing, the provincial programs had to meet the following standards: First, comprehensive coverage had to be provided for all medically required services by physicians with no exceptions and no limits on the amount of coverage provided. The only requirement to receive benefits would be medical need. Second, coverage had to be available on a uniform basis to all eligible residents and provincial programs had to cover at least 95 percent of the population. Third, benefits were to be portable between provinces and during temporary absences from Canada. Finally, the provincial plans were to be administered publicly and accountable to the provincial governments for their financial transactions. Eventually, all ten provinces offered medical care insurance programs.

This then, has been the development of the Comprehensive Hospital and Medical Services Program which exists throughout Canada today. It is a network of provincial systems which have been drawn together by national terms and conditions which were established for federal cost-sharing purposes. The system that has developed is sufficient-

†In 1655, Estienne Bouchard, a Montreal physician, contracted with 26 heads of families to provide medical treatment in return for 100 "sols" per person per year. This was the earliest prepaid health insurance plan in North America. Dr. Bouchard's fee amounted to approximately one dollar per annum. Plague, smallpox, leprosy, epilepsy, and lithotomy were specifically excluded from coverage—*Health Services in Canada*, published by the Department of National Health and Welfare.

ly cohesive so that even recent changes in financial arrangements, and the withdrawal of federally established program conditions, are expected to have little impact on the common features of the various provincial programs.

RECENT GOVERNMENT ACTIVITIES

In more recent years, government activities have been concentrated on filling the gaps and services and extending the range of services available. In Saskatchewan, for example, we now operate a Prescription Drug Plan which has reduced the costs to our residents for high quality, therapeutically effective drug products. This is a universal program in which the consumer pays part of the cost of each prescription and coverage begins with the first prescription. The program is based on a formulary system which currently includes over 1300 drugs. While the existing wholesale and pharmacy system is used for dispensing drugs, the government is involved in purchasing, and has used volume purchases effectively to reduce the costs. Saskatchewan is the only jurisdiction in North America which provides really comprehensive dental care services for children, excepting cosmetic orthodontics. By September 1978 children aged four to eleven years will receive free dental care through an extensive network of clinics located in Saskatchewan schools throughout the province. We have relied heavily upon the skills of dental nurses working under the supervision of dentists who provide preventive, restorative, and surgical services. Other programs dramatically have reduced the cost of hearing aids, and provided orthotic and prosthetic devices free of charge to those who need them. In addition, a wide range of public health and psychiatric services are provided throughout the province.

HEALTH CARE COSTS

Canada, then, has a health care system which ranks among the best in the world. It involves more than 38,000 doctors, 350,000 health care workers, and 1400 hospitals. Proportionately, Canada spends more on health care than any other nation except the United States. Our population is guaranteed a high quality of care with access based solely on medical need. Personal financial circumstances are not a factor in the availability of medical care. In comparison, it is estimated that over 10 million people in the United States lack protection of either public or private hospital insurance. Public funding covers only about 40 percent of the national health care bill and catastrophic illness may result in bankruptcy or drastic changes in family lifestyle. There has been an extremely broad acceptance on the part of the Canadian public of the health care system. A Gallup poll conducted in November 1977 found that 82.6 percent of the people polled expressed a very high satisfaction with the present services.

However, now that we have reached the point where we have an extensive health care system in place, we are turning our attention to the resolution of a couple of major problems. The first is the matter of rapidly escalating costs and second the matter of resource allocation. The rapid development of our health care system over the past two decades has been paralleled by an equally rapid increase in health care expenditures. Since 1960, per capita health care expenditures have averaged a 10 percent annual increase. This includes both private and public expenditures. By 1976, per capita expenditures were estimated at $570 in comparison to a base of $100 per capita in 1960. Health care spending had grown to 7.2 percent of the gross national product by 1975.

What are the reasons for this growth in health care costs? The major factor obviously has been the expansion of services during this period of time. As more services were covered and programs introduced the bill expanded accordingly. In addition, health care workers increasingly have become unionized and have negotiated catch-up wage settlements. They no longer are content to be paid poorly, and consequently improvements in wages dramatically have affected health care costs. Also, the expectations of the general public have increased considerably. They now expect high quality services. And they have come to expect those services fairly close to home, which can be very expensive in sparsely populated areas. This situation, however, has been aggravated by an aging population. Traditionally, the elderly have been the high utilizers of hospital and medical services; I don't think that's news to you. It has been projected that by 1996 the number of Canadians over age 65 will have increased from the present two million to more than three million. For Saskatchewan this aging trend means that by 1985, 28 percent of all medical services and 46 percent of all hospital services will be utilized by senior citizens. Another factor affecting escalating costs is the impact of increased medical specialization. This has been particularly true in the case of chronic and degenerative diseases. The complexity of treating some diseases such as cancer has led to more extensive and intensive forms of treatment.

At the same time, increasingly sophisticated diagnostic and therapeutic equipment has become available. Unfortunately, the technological advances such as CAT scanners carry a heavy price tag, both in terms of purchase price and operating costs. Furthermore, a psychology has developed over the years where we feel that additional resources will continue to have a specified impact. Unfortunately, in most areas we have reached the point of diminishing returns and there is no longer a correlation between resources supplied and improvements in health status. For example, an increase in the supply of physicians has no measurable impact on health status beyond the ratio of 100 physicians per 100,000 population. Physician supply does, however, have a considerable impact on the cost of medical care services.

CHANGES IN HEALTH CARE NEEDS

Finally, health care needs have changed over the past quarter century. The nature of the major diseases has changed to degenerative illnesses such as heart disease and cancer. These diseases are not treated as easily as the major killers of the past such as dysentery, tuberculosis, and pneumonia. Also, we have entered the area of lifestyle-related disease, which has demanded greater attention in recent years. Governments have been faced with the problems of reallocating resources. The public has not been prepared to accept a diversion of funding from a highly popular but costly program. This public attitude has made it extremely difficult politically to redirect monies away from the health system to accommodate changing needs. By the time the provincial governments became deeply involved in the provision of health services, the basic patterns of facilities and resources had been established. In many cases, governments inherited a rather inefficient system of facility distribution. While provincial governments have been able to control the location of new facilities, equipment, and services, little success has been made in rationing the existing base of facilities. A classic example pertains to the duplication of hospital facilities and services. Competition between neigh-

boring communities frequently has resulted in excess capacity and subsequent underutilization of facilities. For example, while the mean hospital occupancy rate in 1975 was 79 percent in one province, the hospitals in the bottom quartile had an occupancy rate of only 55 percent. When you consider the fixed cost associated with staffing a hospital, it is easy to see how this excess capacity contributes to the high cost of health care.

OVERUTILIZATION OF SERVICES

We also are experiencing the overutilization of physician and hospital services when much health care could be managed adequately by general practitioners or paramedics. Similarly, many hospital-based services could be provided on an out-patient or ambulatory basis at a fraction of the present cost. The overutilization of hospitals is an unfortunate side effect of the relatively early insurance of hospital services. As a result of the tendency to use the insured part of the health care system, this pattern has been difficult to deter. This institutional orientation accounts for Canada's high hospital admission rates which are among the highest in the world. One of the most important lessons we have learned in Canada is that once a hospital insurance scheme has become well established it becomes almost impossible to convince people of the benefits of ambulatory or neighborhood projects. Public psychology develops which considers institutional services as the "best" services. And, other forms of service are regarded as second-rate. Therefore, I would caution you to consider carefully the development of ambulatory and community services before the institutional services become entrenched.

SASKATCHEWAN'S APPROACH TO THE PROBLEMS

These then, are some of the major problems which we face in our health care system. You may wonder what approach we have adopted in addressing these issues. I will now speak primarily from the Saskatchewan experiences, since this is the area with which I am most familiar. In recent years, we have placed increasing emphasis upon preventive health measures. For example, the introduction of the dental plan for children is an effort to reduce the incidence of dental disease through the practice and promotion of good dental care. The major factor influencing our decision to enter this field was the shortage of dentists in rural areas. Without a program of this sort, many children simply did not have easy access to adequate dental care. There has been a clear shift in emphasis to heighten public awareness of the effects of lifestyles on health, especially through the use of mass media programs. Such programs in the city of Saskatoon effectively have promoted an increased level of physical activity. In 1974 we introduced the Aware Program which is aimed at developing a public awareness of the implications of alcohol abuse. By encouraging more responsible attitudes toward alcohol use we hope to alleviate some of the social and health problems associated with its abuse. More recently, we have begun a lifestyle program known as "Feeling Good." This program promotes a sensible approach to physical activity, nutrition, smoking, and so on. We think that it will have beneficial effects. In 1977 we passed mandatory seat belt legislation which already has had a significant effect on the incidence of injuries in vehicle mishaps.

While individually these initiatives may appear relatively minor, we hope the cumulative effect will be to reduce the suffering and cost associated with the top four causes of death in Saskatchewan today—heart disease, cancer, strokes, and accidents. We have begun to focus more clearly on the problems of the elderly and we are trying to provide alternatives to institutional care for our senior citizens. An experimental day-hospital currently is being planned in the city of Saskatoon, and changes are being made in the provincial network of home care programs. Some of the alternatives to institutional care may not prove to be less costly options; however, they certainly will improve the quality of life for the elderly. An expanded home care program is not expected to have an immediate impact on the demand for institutional care but we are hopeful that it will eventually postpone or eliminate the entry into nursing homes.

CONCLUSION

Changing public attitudes is a long-term process. However, we in Canada have recognized that the time has come to focus our efforts in this direction. We no longer can accept escalating health care costs. Since reducing access to health care is not an acceptable option we must assume greater responsibility for our health status and a more responsible attitude toward the use of health care services.

In Saskatchewan, we are optimistic about resolving these programs because we have wide support from the health profession. The antagonism which existed when the government first entered the health field has been replaced by a spirit of cooperation. This support is very important in any effort to make adjustments to the health care system. The situation in the United States differs substantially from the Canadian experience. It is generally our perception that the upper levels of health care professionals in the United States are highly rewarded for their efforts. State involvement on a scale comparable to that which exists in Canada would further escalate health costs which already are the highest in the world. Therefore, it would appear extremely difficult for the American Government to implement similar mechanisms to control costs without creating an abrasive relationship with the health care professionals.

I appreciate this opportunity to share the Canadian experience with you and I hope that it may have some bearing on your discussions.

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Breast Cancer Metastatic to Uterine Leiomyoma

ROBERT K. SPIRO, M.D., Bloomfield

The incidence of metastasis of cancer of the breast to leiomyoma of the uterus is low. Two cases of metastasis of a breast malignancy to leiomyoma of the uterus are presented. Discussion concerning the infrequency of this occurrence and a possible explanation is offered.

The incidence of metastasis of cancer of the breast to leiomyoma of the uterus is low. Weingold and Boltuch, in 1961, reported a case of a thirty-eight-year-old female who had a radical mastectomy for scirrous carcinoma of the breast which subsequently metastasized to a uterine leiomyoma. The patient also had irradiation, post-mastectomy. They note six other cases of similar metastatic pattern.[1]

Two patients with metastasis of breast cancer to a leiomyoma are described.

CASE ONE

A patient entered The Mountainside Hospital on 12/7/73 for removal of a mass in the right breast which she had noted six months before. Physical examination was not revealing except for a four cm. mass in the lateral aspect of the right breast. The mass was somewhat firm and there was some skin retraction. Laboratory studies revealed 10.7 grams percent of hemoglobin. The SMA 12, chest x-ray, and bone scan were negative. The past history was not unusual. A benign cyst had been removed from the right breast in 1964.

A modified radical mastectomy was performed and a partially differentiated infiltrating adenocarcinoma of the breast with metastases to the lymph glands was found (Figure 1). Following surgery the patient received 4,600 rads using the linear accelerator in 22 treatments between 3/8/74 and 4/10/74. The therapy was directed to the chest wall and the breast drainage area.

She recovered but in August, 1976, complained of nausea, retching after meals, and a tendency to fatigue easily. She continued to menstruate. Hemoglobin was 8.7 grams/dl. Chest x-ray showed some pulmonary fibrosis secondary to irradiation. The bone scan showed radioactive activity in the ribs, dorsal and lumbar spines, and possibly left hip. A mass was noted on pelvic examination and confirmed by intravenous pyelogram. The anemia was corrected before surgery. At operation a large uterine leiomyoma was found, so total hysterectomy and bilateral salpingo-oophorectomy were done. Pathological report revealed metastatic breast adenocarcinoma in the uterine leiomyoma (Figure 2).

A soft, two cm. mass appeared in the right vaginal vault in April 1977. It was removed and was diagnosed as a leiomyoma of the vaginal wall. The patient continues to prosper and her bone scan has returned to normal.

CASE TWO:

A forty-six year old female entered The Mountainside Hospital on 1/22/76 because of a right breast mass and a pelvic mass. She had known of the breast mass for several weeks.

Examination revealed an ill-defined, three cm. tumor

*From the Department of Surgery, The Mountainside Hospital, Montclair. Dr. Spiro may be addressed at 1218 Broad Street, Bloomfield, New Jersey 07003.

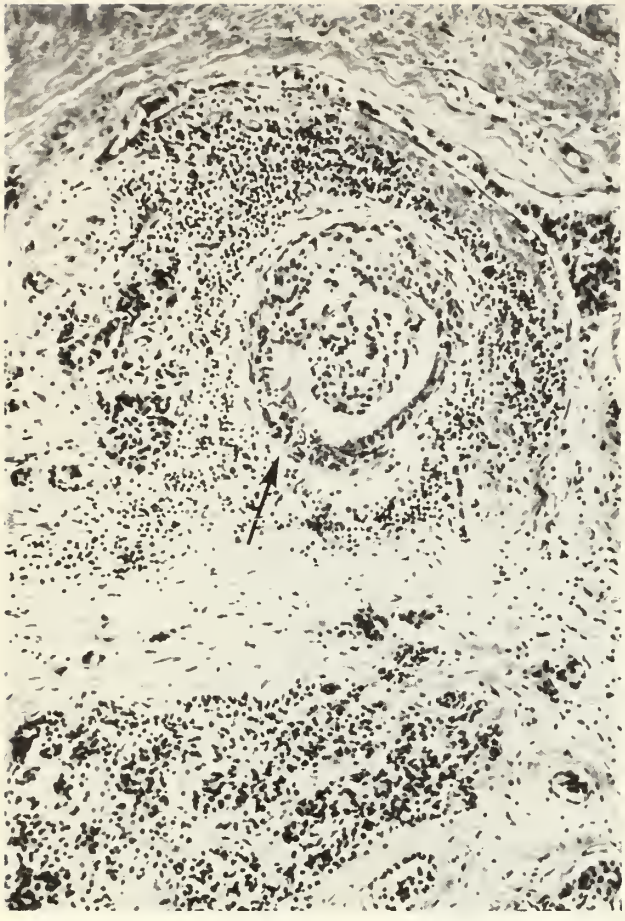


Figure 1—Adenocarcinoma of the breast. Malignant cells are seen within a duct. (Case 1)

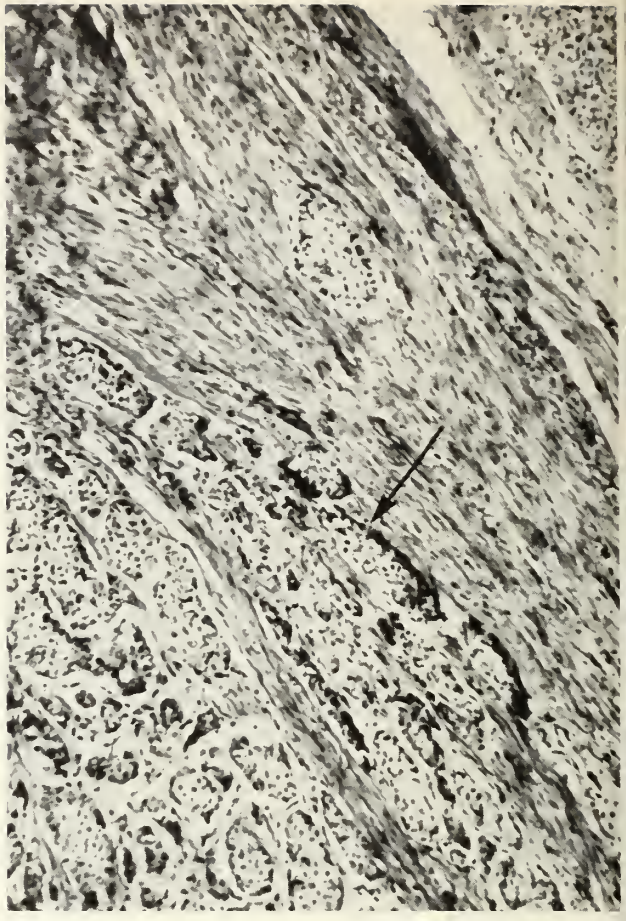


Figure 2—Adenocarcinoma of breast metastatic to uterine leiomyoma. Tumor cells are seen within muscle sheets. (Case 2)

lateral to the right areola. The pelvic mass was round and occupied the right pelvis. Laboratory data including bone, brain, and liver scans, and chest x-ray were normal.

Surgery was performed the next day and a partial mastectomy was done for adenocarcinoma of the breast (Figure 3). Abdominal exploration revealed many small tumor nodules in the liver, the largest being one cm. in diameter. A 10 cm. intraligamentous leiomyoma was found in the right broad ligament. The leiomyoma and bilateral salpingo-oophorectomy were surgically removed. Pathological reports revealed poorly differentiated adenocarcinoma of the breast (Figure 3), with metastases in the liver and leiomyoma of the uterus (Figure 4). There was moderate cystic hyperplasia of the endometrium. The ovaries had no pathologiccal diagnosis. The patient recovered from surgery well and was treated with 5 fluorouracil and thiotepa and methotrexate.

In November 1976, she was readmitted to the hospital for pancytopenia and mucosal bleeding ascribed to her cytotoxic therapy. She had been receiving thiotepa and methotrexate. Liver and bone scans were abnormal. Blood was administered; she improved and was discharged to the continuing care of her oncologist.

Two months later she was hospitalized again for weakness; anorexia, and vomiting. Hypercalcemia was present. She was treated with parenteral fluids, steroids, and mithramycin.

The calcium did decline toward normal. However, she progressively grew worse and died, approximately one year after surgery. Permission for autopsy was not granted.

DISCUSSION

The finding of metastasis from one tumor to another is uncommon. Virchow, according to Weingold and Boltuch[1] felt that organs frequently beset by primary tumors were seldom involved with metastases. The uterus appears to fit his thesis. It is well known, however, that when tumor cells enter the blood stream, emboli can lodge in odd places and this may explain present circumstances.

Almost 70 percent of the malignancies occurring in the female genital tract in the United States are in the uterus.[2] Metastasis from one pelvic genital organ to another is believed to be primarily lymphatic or transtubal. Direct extension, of course, occurs.

The reason for localization of metastases is unknown. Willis stated: "Particular tissues provide optimal or chemical metabolic conditions with growth of particular tumours; different tumours have different optimal requirements therefore each tumour has its own preferential site of metastases."[3]

It has been thought that the relationship of the main arterial source to an organ to its immediate proximal vascular origin, is significant. If the relationship is somewhat like a right angle, as is the case of the hypogastric artery to the common ilian, the malignant cells might flow by the ostium of the hypogastric artery, and thus not reach the uterus. Metastasis to muscle is unusual, perhaps because the contraction of the muscle inhibits the lodgement and growth

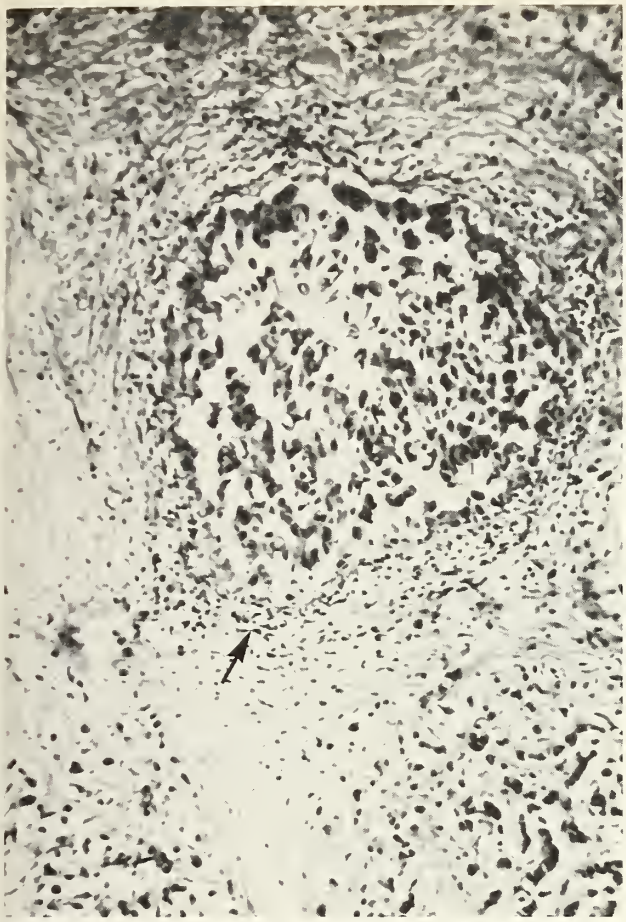


Figure 3—Adenocarcinoma of breast. Tumor cells are seen within a duct. (Case 2)

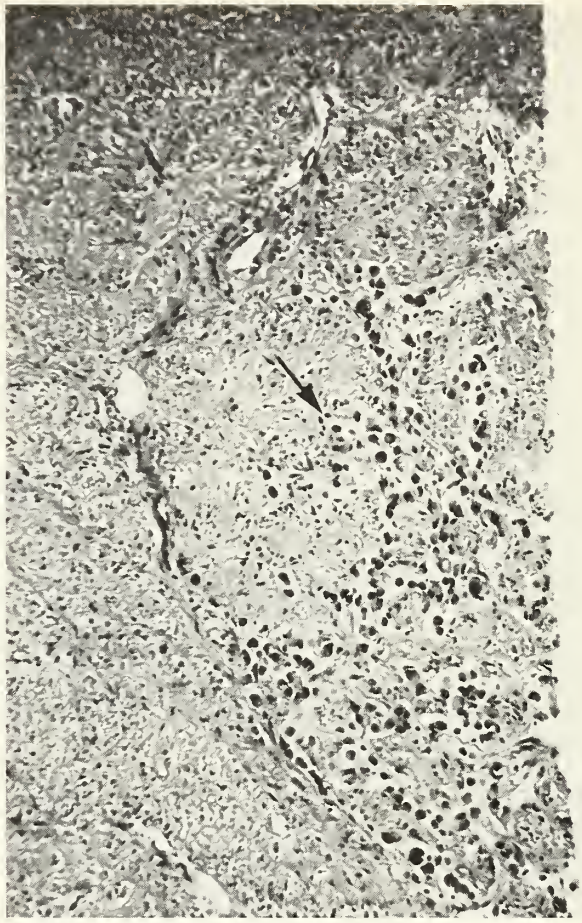


Figure 4—Adenocarcinoma of breast metastatic to uterine leiomyoma. Malignant cells are seen within muscle sheets. (Case 2)

of tumor cells. Metastasis to the uterus usually occurs to the endometrium rather than myometrium.[1]

It may be wise to consider a total hysterectomy and bilateral salpingo-oophorectomy, if castration is being done for breast carcinoma and a leiomyoma of the uterus is encountered. This obviously would remove a focus of metastatic cancer that customarily might be left behind. It is acknowledged, however, that the incidence of metastasis from breast cancer to a uterine leiomyoma is rather uncommon.

SUMMARY

Two cases of metastasis of a breast malignancy to leiomyoma of the uterus are presented. Discussion concerning the infrequency of this occurrence and a possible explanation is offered.

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REVIEW ARTICLE

The Surgical Management of Abdominal Trauma

WILLIAM A. DWYER, JR., M.D., Paterson*

This paper presents a review of the current literature on the diagnosis and management of abdominal trauma. The diagnostic and surgical management aspects are considered separately and the points of strength and weakness in each of these areas are considered.

The management of the seriously injured patient is, at all times, a surgical challenge. There is a need first to ensure an adequate airway, then to restore volume losses, splint fractures, and finally to assess the patient in terms of the necessary surgical repairs to restore his homeostasis.

All too frequently, it is necessary to carry out all of these measures virtually at the same time. Given this sort of time frame and the multiplicity of organ systems that may be involved, it is quite conceivable that mistakes can be made and relatively occult organ injuries overlooked.

This is particularly true of the contents of the abdominal cavity which are not visualized easily except by special x-ray techniques and in which damage can remain obscure for a number of hours or even days before its disastrous consequences become obvious. It is not surprising, then, to find reports indicating that abdominal injuries account for 41 percent of all deaths in patients in whom there were combined injuries (head and abdomen, chest and abdomen, and so on).[1,2]

It should be borne in mind that this figure represents those trauma victims who reached the hospital alive. As our emergency medical services improve under the impetus of federal grants, the numbers of patients, severity of injury, and mortality rate will increase unless the surgical management of these cases also improves. This point is well illustrated by the work of Fitzgerald, Crawford, and DeBakey who found that 75 percent of patients with blunt abdominal trauma in Houston, Texas died prior to reaching the hospital.[3] Stevenson, in his report on the experiences in Belfast, commented on the efficiency of the ambulance services and the concomitant increase in severity of the wounds seen.[4]

The patient with a traumatized abdomen presents the surgeon with both a diagnostic and a therapeutic challenge.

DIAGNOSIS

The initial and most important question is whether or not significant hemorrhage or compromise of organ integrity has occurred within the abdominal cavity. This question frequently is made more complex by the existence of injuries to other organ systems. Particular note should be taken of the possibility of abdominal organ involvement where a penetrating wound of the chest has occurred. Danto *et al.*, in their study of penetrating torso injuries, noted that seven of 27 cases of thoracic gun-shot and 12 of 37 stab wounds had significant intra-abdominal organ injury.[5]

History alone is usually not of much assistance in the diagnosis and management of the trauma case. The kind of instrument or the kind and amount of force involved in causing the injury may heighten clinical suspicions in certain cases.

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By contrast, physical findings are of immense value. Shaftan made the point that significant intraperitoneal injury results in clinical findings that are recognized easily.[6] Nance, writing fourteen years later, came to the same conclusion after reviewing the New Orleans Charity Hospital experience with more than 2000 cases.[7] Shaftan's remark in discussing Nance's paper is worth quoting:

"I feel it is reasonable . . . to make a plea for not treating civilian penetrating injuries of the abdomen, even gunshot injuries, by dogma or dictum. We agree entirely with Dr. Nance, and other authors, that gunshot wounds are comparatively easy to evaluate clinically, and that the decision to operate can be made rapidly and accurately on these grounds alone."

The existence of a penetrating wound obviously raises concern that all may not be well within the abdominal cavity. Some attention should be paid to the presence of both entrance and exit wounds as well as the possible existence and pattern of bruises of the torso. Abdominal distension, particularly in the early phases of treatment, can be indicative of the presence of fluid or air in the abdominal cavity.

While tenderness to palpation may or may not indicate significant intra-abdominal problems, the presence of diffuse rigidity and rebound tenderness is an unequivocal indicator of the need for urgent laparotomy.

While normal blood pressure does not rule out major blood loss, hypotension does indicate significant blood volume loss. Tachycardia may indicate major problems, but a normal pulse rate does not exclude them. Finally, the initial absence of bowel sounds or their subsequent disappearance is also highly suggestive of intra-abdominal injury.

The usual laboratory parameters are of some assistance in diagnosis of intra-abdominal injury, more particularly when used in the course of observation of the trauma patient than in primary evaluation. Most such patients, even in the face of major hemorrhage, have a normal hemoglobin on admission. Serial hemoglobin levels at four- to six-hour intervals that show a progressive decline speak for significant bleeding. Most patients without serious injury should have a normal or only slightly elevated white blood cell count. If peritonitis is developing, or if there has been injury to liver or spleen, the white count should show an initial elevation or a progressive elevation over time.

The urine should be examined both grossly and microscopically for evidences of blood.

Serum amylase may be of value. This test invariably is elevated in patients with pancreatic injury and may be elevated where rupture of stomach, duodenum, or small bowel has occurred.

X-rays also can be helpful, if the general condition of the patient permits. There is no need for x-rays in the patient in profound shock with a single bullet wound of the abdomen. Such a patient requires rapid volume replacement and urgent laparotomy.

Where definite clinical indications for surgery do not exist immediately, some x-ray studies can be of assistance. The easiest to obtain, and probably the most valuable, is the upright chest. This film may show air under the diaphragm indicating a ruptured viscus, an air-filled viscus above the diaphragm indicating diaphragmatic rupture, or fractured ribs which may be a clue to splenic or hepatic damage. Marked right-sided elevation is indicative of herniation of the liver through a right-sided diaphragmatic rupture. Pneumothorax, mediastinal shift, and widened mediastinum, which speak for possible rupture of the thoracic aorta, and fluid in the chest are also diagnostic benefits to be gained from such an x-ray.

Flat films of the abdomen are of marginal value, but may indicate splenic enlargement or downward displacement of the air bubble in the splenic flexure by an enlarging spleen. It also may show air in the retroperitoneal space from a rupture of the duodenum or the retroperitoneal portions of the colon. Either an upright or lateral decubitus film of the abdomen also should be taken if the patient's condition permits. This, too, may show evidence of extra-luminal air which was not evident on the flat film.

Intravenous urography is of value in all patients with hematuria. It can show damage to the kidney, but also ureteral rupture, and in some cases, displacement of the ureter by retroperitoneal hematoma.

Selective arteriography is useful in certain cases. This technique can pinpoint bleeding or injury to specific organs such as kidney or spleen with reasonable accuracy.[8] Where hematobilia is suspected, a splenoportogram is helpful.[9] Injections of the wound with opaque dye have not proved to be of major value, in view of the number of false negatives.[10]

Isotopic scan studies of the liver and spleen may show damage to these organs in selected cases.

Of all the adjunctive maneuvers currently available, the most productive appears to be abdominal paracentesis or lavage. Davis cited an accuracy rate of 86 percent for paracentesis utilizing a Potter side-hole needle.[1] Danto, in his review of the evaluation of the abdomen in penetrating torso injuries, cited an accuracy rate of over 70 percent with no false negatives and five false positives.[5] As contrasted with a control group of similar patients, he found that those evaluated by paracentesis had a negative celiotomy rate of 8.6 percent as compared to a 21 percent negative celiotomy rate for those evaluated without its use. By contrast, Jordan and Beall cited both false negative and positive results utilizing the four-quadrant abdominal tap and advocated the use of peritoneal lavage as espoused by Gumbert, and modified by them.[10,11] In their hands, a perfusate that is colored pink should be considered an indication for laparotomy. They additionally advocate an amylase determination on the perfusate and, if the fluid is turbid, a microscopic cell count.

It should be emphasized that the technique of paracentesis does not supplant clinical evaluation. It is most useful, in those patients who present with no initial clinical findings, to help determine the need for urgent operation.

Table I summarizes the aids available to the surgeon with which he can arrive at a firm conclusion as to the necessity of urgent operation in abdominal trauma.

This may seem to some a very limited diagnostic effort but, given the seriousness of many of these problems and the time constraints imposed by the critical status of the patients, this is a very practical approach to the problem. The alterations in physiology imposed by the traumatic insult pose problems for the patient. The imposition of additional trauma in the form of a surgical exploration represents a considerable further burden to many patients. Originally Shaftan,[6] and subsequently other authors reported on the advantages of selecting patients for either urgent operation or careful observation based on a system of indicators (principally clinical findings) for laparotomy. Nance, reporting on his experience, indicated a reduction in morbidity of 15 percent following adoption of such a policy.[7] Forde *et al.*,[13] reporting on 610 cases of penetrating abdominal trauma treated under a policy of mandatory exploration for penetrating wounds,

Table 1
Indications for Urgent Laparotomy

I History — Gunshot, shotgun or implement wound involving abdominal cavity

II Physical Examination
- Penetrating wound (gunshot) through or in proximity to peritoneal cavity
- Stab wounds with evidence of
 - a) Peritonitis (abdominal tenderness, guarding, diminished bowel sounds, fever)
 - b) Signs of bleeding (unexplained hypotension; tachycardia; hypovolemia; blood in rectum, nasogastric aspirate, or urine)
 - c) Evidence of evisceration
 - d) Evidence of injury to diaphragm

III Paracentesis
- a) Evidence of blood in abdominal cavity
- b) Evidence of turbid fluid with positive cell count
- c) Fluid in abdominal cavity with high amylase

IV X-ray Studies
- Flat plate and upright films of abdomen, P-A chest
 - a) Free air in abdominal cavity
 - b) Air in retroperitoneal space
- Cystogram
 - a) Rupture of bladder
- Drip nephrotomogram (indicated in presence of hematuria)
 - a) Damage to kidney
 - b) Displacement or disruption of ureter
 - c) Damage to bladder
- Arteriogram
 - a) Positive evidence of damage to organs
 - b) Positive evidence of bleeding from arterial bed
- Radioisotopic studies of liver and spleen
 - a) Can show positive evidence of organ damage

found a negative exploration rate of 39 percent for stab wounds and 20 percent for gunshot wounds. The complication rate for the group undergoing negative exploration was 8.7 percent. For these reasons, the diagnosis of intra-abdominal injury should be firm prior to undertaking surgery.

SURGICAL MANAGEMENT OF SPECIFIC ORGAN INJURY

Firm evidence of intra-abdominal injury warranting celiotomy, however, still leaves a considerable surgical challenge. Stab wounds frequently involve only one organ. Gunshot wounds, particularly the low muzzle velocity wounds seen in civilian practice, also can be associated with a limited pathological state. Blunt trauma, on the other hand, poses a considerable diagnostic problem and can be associated with a number of occult pathological states. Of particular importance, in this regard, are injuries to the duodenum and pancreas, injuries to the gut and its mesentery which result in delayed rupture, and occult intra-peritoneal or retroperitoneal bleeding.

The surgeon thus is faced with the necessity to move rapidly and thoroughly in his surgical examination of the abdominal cavity and to consider the possible existence of those lesions that tend to remain occult in that examination. Table 2 represents an analysis of three reports on treatment of blunt trauma and indicates the relative frequency of each organ involvement in each series.[1,2,14] It readily can be appreciated from this table that, while the liver and spleen are the organs most frequently involved, there is no abdominal organ that is immune from involvement, including the diaphragm.

The management of injuries of the spleen has undergone little change over the years; classically, it consists of splenectomy. While this is still a necessity for major organ injury, there are an increasing number of reports dealing with both suture of the splenic capsule and the use of Avitene®† in cases involving lesser degrees of damage.[14] With the advent of knowledge regarding the spleen's role in the immunological process, particularly in children, this more consevative approach seems to be warranted wherever possible.

Hepatic injuries always have been a surgical challenge. All current authors on the subject condemn the use of packing and advocate the technique of effecting hemostasis without approximation of liver tissue in simple wounds of the liver.[16] As far as can be ascertained from the literature, all such wounds should be drained utilizing big drains. Where major fractures of liver parenchyma exist, either a formal hepatectomy or dissection of the liver utilizing the "finger fracture" method of dissection, are the procedures of choice at this time. (Again, extensive drains should be inserted prior to closure.) There have been some reports regarding the efficacy of ligating the hepatic artery in injuries of the liver to control bleeding.[15] However, this procedure must be considered as one still under evaluation. Previous work indicating a beneficial effect of T-tube drainage in hepatic injury has not been substantiated by other investigators and is used only in cases involving major bile-duct injury.[11] Even in the best of hands, major injury to the liver is associated with a high morbidity and mortality. One series reports 12 percent mortality with an additional 20 percent morbidity, which roughly approximates that in other reported series.[10]

Small bowel injuries are handled in the conventional manner with resection of damaged bowel (and mesentery) and end-to-end anastomosis. Large bowel injuries have been a subject of controversy for some years. Most surgeons have been trained to exteriorize all such wounds wherever pos-

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Table 2
Frequency of Abdominal Organ Involvement in Blunt Trauma

Davis – 437 Cases	Stoppo – 168 Cases	Balton – 59 Cases
Spleen	Spleen	Liver
Liver	Liver	Spleen
Jejunum, Ileum	Kidney	Kidney
Kidney	Mesentery	Diophragm
Blodder	Bladder	Small bowel
Mesentery	Vasculor injuries	Large bowel
Right colon	Small bowel	Mesentery
Left calon	Lorge bowel	Boll blodder
Poncreas	Poncreos ond Duadenum	Urinary blodder
Duodenum	Stomoch	Poncreas
Goll blodder	Adnexo	Stomoch
Diaphrogm		Aorto
Stomoch		

sible. Given a military situation, this appears to be the procedure of choice. However, in the civilian type of injury commonly seen and granting a minimal soilage of the peritoneum by feces, these wounds can be managed by debridement of devitalized tissue and a two-layer closure of the bowel with good results.[16] Proximal colostomy coupled with primary repair is considered as the prodecure of choice in wounds of the recto-sigmoid area below the peritoneal reflection.

Injury to the pancreas is an infrequent surgical finding because of its protected location. However, its incidence is increasing. It is rarely an isolated injury and should be surgically managed after the completion of other necessary procedures. Contusions should be drained. Lacerations should be repaired and drained. Devitalized pancreas should be resected en bloc. Management of duct injury requires transduodenal pancreatography to define the involvement, resection of the devitalized portion of pancreas and Roux-en-Y anastomosis of small bowel to the remaining pancreas for drainage purposes. In rare cases of extensive duodenal and pancreatic injury, a Whipple operation may be necessary.

Major vessel injury is probably the most lethal of the many problems involved in the management of abdominal injury. Progressive improvement in the mortality rate is a function of the remarkable instrumentation and techniques developed in recent years by the vascular surgeons and of the improvements in ambulance service. Bleeding from the aorta or vena cava first should be controlled by direct pressure with a finger or a pack. Only then should definitive control be attempted. In the case of extensive aortic lacerations or multiple arterial injuries, it is sometimes helpful to occlude the aorta at the diaphragm. The use of an intracaval shunt has simplified the management of suprarenal vena cava injuries, although this continues to be a very lethal injury.[17] The artery most commonly injured, according to Beall and Jordan is the renal artery; they believe repair is frequently possible with salvage of the kidney.[10]

Injuries to the gall bladder are rare and usually simply are managed by cholecystectomy, but injury to the bile ducts can be quite trying. Injuries above the bifurcation should be treated by one layer closure without a T-tube stint; in common bile duct injuries a T-tube is advisable. In all instances, drainage is advocated.

CONCLUSION

Diagnosis and management of patients with abdominal trauma always has been a serious surgical problem. There has been an increasing number of cases involving blunt abdominal trauma. It is predicted that this will continue and the associated injuries will increase in severity. Under the influence of Shaftan, Nance and others, greater emphasis is being placed on selecting cases for operation subject to penetrating trauma rather than operating on them categorically. The primary basis for such selection is the patient's clinical findings, initially or as they develop in the course of progressive observation. Certain additional clinical and laboratory tests are mentioned which aid in this selection process. Newer techniques in the surgical management of specific organ injuries also are discussed.

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213th Annual Meeting
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DAILY SCHEDULE

Friday, May 11, 1979
3:30 p.m. —Board of Trustees

Saturday, May 12, 1979
8:00 a.m. —Registration Opens
9:00 a.m. —Message Center Opens
11:30 a.m. —Meeting—NJ Committee on Trauma
11:30 a.m. —Luncheon—NJ Orthopaedic Society
12 noon —Scientific and Informational Exhibits Open
12 noon —Luncheon—NJ Committee on Trauma
1:00 p.m. —Annual Spencer T. Snedecor Trauma Oration
2:00 p.m. —Motion Picture Theatre
2:00 p.m. —House of Delegates
3:30 p.m. —Reference Committees: Constitution and Bylaws, "A", "B", "D", "G"
6:00 p.m. —Reception for Alumni—Jefferson Medical College

Sunday, May 13, 1979
7:15 a.m. —Annual Prayer Breakfast
8:00 a.m. —Registration Opens
8:00 a.m. —Scientific Session on Oncology
9:00 a.m. —Message Center Opens
9:00 a.m. —Scientific and Informational Exhibits Open
9:00 a.m. —Scientific Sessions:
Allergy, Emergency Medicine, Medicine
Dermatology, Obstetrics and Gynecology
Family Practice, Pediatrics
Neurosurgery and Neurology
Psychiatry
9:30 a.m. —Meeting—NJ Academy of Ophthalmology and Otolaryngology
10:00 a.m. —Motion Picture Theatre
10:00 a.m. —Reference Committees: "C", "E", "F", "H"
11:30 a.m. —Luncheon—NJ Academy of Ophthalmology and Otolaryngology
12 noon—Luncheon—NJ State Urologic Society
12:15 p.m. —Luncheon—NJ Chapter, American College of Emergency Medicine
1:00 p.m. —Luncheon—Oncology Society of NJ
1:00 p.m. —Scientific Sessions:
Anesthesiology
Cardiovascular Diseases, Medicine, Nuclear Medicine
Ophthalmology
Otolaryngology
Plastic and Reconstructive Surgery
Urology
2:00 p.m. —Motion Picture Theatre
3:00 p.m. —House of Delegates
4:30 p.m. —Meeting—Widows and Orphans Society
6:30 p.m. —Inaugural Reception Honoring President-Elect and Mrs. Alessi
Hosts—MSNJ and Bergen County Medical Society
All members, official guests, and their wives, and Auxiliary members are cordially invited
—Tickets $5.00 per person
8:00 p.m. —Inaugural Dinner
Hosts—MSNJ and Bergen County Medical Society
Music and Entertainment—Danny Davis and His Nashville Brass
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Monday, May 14, 1979
7:00 a.m. —JEMPAC Breakfast
8:00 a.m. —Scientific Session on Orthopaedic Surgery, Family Practice
8:30 a.m. —Registration Opens
8:30 a.m. —Scientific Session on Surgery, Clinical Pathology
9:00 a.m. —Scientific Sessions:
Medicine, Rheumatism
Radiology
9:00 a.m. —Message Center Opens
9:00 a.m. —Scientific and Informational Exhibits Open
9:00 a.m. —JEMPAC Political Action Seminar
9:30 a.m. —Scientific Session on Chest Diseases
10:00 a.m. —Motion Picture Theatre
11:30 a.m. —Luncheon—NJ Society of Physical Medicine and Rehabilitation
12:00 noon —Luncheons:
NJ Chapter, American College of Chest Physicians
NJ Orthopaedic Society
1:00 p.m. —Scientific Sessions:
Gastroenterology and Proctology
Physical Medicine and Rehabilitation
1:00 p.m. —Meeting—NJ Foundation for Health Care
2:00 p.m. —Motion Picture Theatre
3:00 p.m. —House of Delegates
3:00 p.m. —Scientific and Informational Exhibits Close
6:00 p.m. —JEMPAC Wine and Cheese Reception

Tuesday, May 15, 1979
6:45 a.m. —Breakfast Meeting of the "Coalition of Southern NJ County Medical Societies"
Reservations: Mrs. Ruth E. Bunning, Exec. Sec., Burlington County Medical Society, P.O. Box 56, Medford, NJ 08055
7:15 a.m. —Breakfast Meeting—Bergen and Passaic County Medical Societies
Reservations: Mr. George Willis, Exec. Dir., Bergen County Medical Society, 170 Main St., Hackensack, NJ 07601
7:15 a.m. —Breakfast Meeting—Essex County Medical Society
Reservations: Mr. Arthur R. Ellenberger, Exec. Sec., Essex County Medical Society, 144 So. Harrison St., E. Orange, NJ 07018
8:30 a.m. —Registration Opens
9:00 a.m. —House of Delegates
12 noon —Registration Closes
2:00 p.m. —Annual Meeting—Board of Governors of the Medical Inter-Insurance Exchange of NJ
3:00 p.m. —Board of Trustees

PEDIATRIC BRIEFS

Selected Abstracts with Comments*

Grommets and Glue Ear: A Five-Year Follow-Up of a Controlled Trial. Brown MJKM *et al. J Royal Soc. Med* 71:353 (1978)

Fifty-five children with bilateral glue ears were included in a control trial in which one ear was drained and a grommet inserted and the other left untreated. The children were observed repeatedly and objectively. Although there was significant improvement in hearing in the first few months, by six months after operation there were no differences in hearing or in acoustic impedance. However, at five years the ears that had grommets inserted had significant increase in tympanosclerosis and scarring. The advantages of short-term hearing improvement must be weighed against the long-term disadvantages of damage to the ear drum and middle ear.

Comment: We need many more studies of this type to make an assessment of the value of myringotomy and tube insertion in the treatment of serous otitis. Until these studies are completed we must be cautious. Therapies are always double edged: cost versus benefit. The costs of myringotomy/tube insertion may be too high to pay.

(R.H. Rapkin, M.D.)

Comparison of Nasotracheal Intubation and Tracheostomy in Management of Acute Epiglottitis. Oh TH *et al. Anesthesiol* 46:214 (1977)

Acute Epiglottitis in Children: Management of 27 Consecutive Cases with Nasotracheal Intubation with Special Emphasis on Anesthetic Considerations. Blanc VF *et al. Can Anesth Soc J* 24:1 (1977)

Comment: These two articles describe the modern management of epiglottitis and protocols are given for treatment of same. These protocols are applicable to community hospitals and easily can be arranged for local situations. Pediatric departments ought to get these references and set up a plan for management of this acute emergency.

(R.H. Rapkin, M.D.)

Cows' Milk Protein Sensitive Enteropathy. Iynkaran N *et al. Arch Dis Child.* 53:20 (1978)

Fifteen patients with histologically proved cows' milk protein enteropathy were studied. Criteria for the diagnosis were developed including clinical disease on cows' milk, clinical improvement on milk-free diet, normal jejunal histology six to eight weeks after removal of milk, histologic relapse on exposure to cows' milk protein. Earlier criteria specifying repeated challenge and removal of offending milk (at least three cycles) often are ignored by the clinician who may overdiagnose on the basis of a single challenge after elimination. Differentiating temporary (or permanent) lactose intolerance may be difficult. Intercurrent infectious gastroenteritis may confuse.

Comment: Manipulation of formulae often has been considered the stock and trade of the pediatrician. Manipulation when carried to extremes is often detrimental. Most "milk allergy" is overdiagnosed. Those patients with continuing malabsorption problems need careful assessment, not trial and error manipulation.

(R.H. Rapkin, M.D.)

Comparison of Sucrose with Glucose in Oral Therapy of Infant Diarrhea. Nalin DR *et al. Lancet* 2:277 (1978)

Oral Hydration in Rotavirus Diarrhea: A Double-Blind Comparison of Sucrose with Glucose Electrolyte Solution. Sack DA *et al. Lancet* 2:280 (1978)

Oral glucose-electrolyte solutions have proved effective in rehydration in patients with cholera, enterotoxigenic E. coli diarrhea and other acute gastroenteritis. Glucose facilitates transport of sodium and water in patients with diarrhea. Sucrose has been used as well and appears effective. Children with various etiologies of diarrhea (including rotavirus) were studied and although glucose was somewhat more effective than sucrose, patients with 5 to 10 percent dehydration were effectively rehydrated orally. Time to recovery and success rate was as good as with intravenous hydration. Stool fluid losses were no greater with oral than intravenous rehydration. Fluids used contained sodium (90 meq/L), potassium (20 meq/L), (chloride 80 meq/L) and bicarbonate (30 meq/L). The concentration of sucrose was 40 grams/L and that of glucose was 20 grams/L. Additional fluids included water and in some, breast milk. Most patients were able to be discharged in 48 hours.

Comment: Oral therapy for diarrhea is practical and effective and the decision to institute intravenous therapy need not be made unless the child is very dehydrated, if appropriate fluids are offered and the child is observed carefully. The use of prepackaged and prepared solutions for outpatient therapy is far safer than homemade solutions. Elimination of cow milk is often wise, perhaps because of lactose but certainly because of its osmolar load. Although

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these studies have far more importance in countries where diarrhea is a major scourge and equipment for intravenous therapy may be unavailable, they have applicability to our milieu.

(R.H. Rapkin, M.D.)

Effectiveness of Neonatal Screening for Congenital Dislocation of the Hip. Place MM *et al. Lancet* 2:249 (1978)

All children with congenital hip dislocation (CH) diagnosed *after* three months of age were studied and compared with children diagnosed in the neonatal period. Twenty-four of thirty-two late-diagnosed patients were examined in the newborn period; 15 were passed as normal; 9 were equivocal but follow-up examination was said to be normal; 16 patients were diagnosed by one year and another 11 by age two. The authors conclude that there is substantial false-negative rate of newborn testing and repeated testing is necessary.

Comment: No surprise here. But, this study does appropriately reinforce previously known data. Newborn exams usually detect dislocatable hips which often self-correct and which can be treated conservatively with a simple abductor splinting. Those hips already dislocated (significant morbidity) may be missed for reasons not clear but perhaps because there is no abduction limitation in these children in the newborn period (Relaxin) and because the hip is "out" there are no clicks. Vigilance and retesting is clearly the answer since those hips do eventually (several months) develop abduction limitation.

(R.H. Rapkin, M.D.)

Long-Term Follow-Up of Preterm Infants in Breech Presentation Delivered by Cesarian Section. Ingemarsson I *et al Lancet* 2:172 (1978)

Outcome for breech presentations delivered before 37th week in Lund, Sweden, was compared in two groups: 42 infants delivered by cesarian section; 48 by vaginal route. The group delivered by section had two deaths and one long-term sequel; by vaginal route there were seven deaths and ten sequelae. The results suggest section as the safer course.

Comment: The authors note a caveat: different time periods were studied and patients were not randomized. However, the results are impressive and, pending confirmation, suggest a rational procedure in this group of patients.

(R.H. Rapkin, M.D.)

Ineffectiveness of Topical Ether for the Treatment of Genital Herpes Simplex Infection. Corey L *et al. N Engl J Med* 299:237 (1978)

Topical ether was ineffective in this disease as has been shown for topical adenine arabinoside, topical idoxuridine, and photodynamic dyes. "For the present our medical armamentarium for mucocutaneous genital infection due to herpes simplex virus consists of accurate diagnosis and explanation of the clinical course, complications and potential of transmission of the disease. . ."

Bronchopulmonary Infection Due to B Lactamase Producing B. Catarrhalis Treated with Amoxycillin/Clavulanic Acid. Minane G *et al. Lancet* 2:257 (1978)

Clavulanic acid inhibits B lactamase and shows synergy with other B lactam antibiotics (e.g. Ampicillin) that are usually inhibited by bacteria which produce the enzyme. In an uncontrolled study (report) these authors demonstrate effectiveness of this combination in treating infection by a B lactamase producing organism.

Comment: Other clinical trials with clavulanic acid suggest optimism to this agent's value. The future of treatment of B lactamase producing H. influenzae should be watched with interest.

(R.H. Rapkin, M.D.)

Bicarbonate Therapy Revisited. Eidelman AU *et al. Am J Dis Child* 132:847 (1978)

Comment: Another attack on the use of bicarbonate in the neonate! The authors summarize a number of studies which looked at the effect of bicarbonate on improving pH and survival in acidotic neonates *after* resuscitation and come to the conclusion that it is of little help and its use therefore should be limited to severe and persistent acidosis when the pH is below 7.15.

I believe that their analysis is basically correct in that most metabolic acidosis in neonates is due to hypoxia and hypoperfusion (shock) and that the proper treatment of such acidosis is treatment of the hypoxia or hypoperfusion. If this is done, only occasionally will there be need to consider the use of bicarbonate. However, in these occasional cases, I would not let the pH fall to 7.15 before resorting to buffer; rather, I would consider using bicarbonate at the pH of 7.20.

The authors also are negative about the use of bicarbonate *during* resuscitation but don't have much on which to base this negative assessment. For example, they don't cite any controlled studies showing a lack of efficacy. Indeed, the one study quoted is that on monkeys performed by Dawes who found that hypertonic bicarbonate sustained cardiorespiratory function during asphyxia.

I would continue to employ bicarbonate in neonatal resuscitation but only after suctioning, stimulation, and bagging with 100 percent oxygen have proved ineffective. The authors' recommendation that one employ isotonic bicarbonate instead of hypertonic bicarbonate seems reasonable as it may limit the incidence of intraventricular hemorrhage and will result in a greater volume of fluids given during resuscitation.

(R. Meny, M.D.)

Neuropathy Following Abuse of Nitrous Oxide. Layzer RB *et al. Neurol* 28:504 (1978)

Polyneuropathy for Inhalation of N_2O Cartridges Through a Whipped-cream Dispenser. Sahenk Z *et al. Neurol* 28:485 (1978)

Acute and Chronic Progressive Encephalopathy Due to Gasoline Sniffing. Valpey R *et al. Neurol* 28:507 (1978)

Comment: Nitrous oxide has become more popular in late adolescents searching for "highs." We need to be aware of this complication, and as part of our medical history, ask about the use of nonprescription drugs. We also need to be concerned about medical personnel who have easy access to nitrous oxide.

(J. Tarnow, M.D.)

Dextroamphetamine: Cognitive and Behavioral Effects in Normal Prepubertal Boys. Rapoport J. *Science* 199:560 (1978)

A single dose of dextroamphetamine (0.5 mgs./kg.) was given to 14 normal prepubertal boys in a double-blind, crossover study. The drug was associated with a marked decrease in motor activity and reaction time, plus improved behavior on cognitive tests. "Rebound" hyperactivity occurred five hours afterwards. Because of the similarity of response of normal and hyperactive boys to amphetamines some doubt is

cast on the clinically specific "paradoxical" effects of amphetamines.

Comment: Amphetamine administration to children is not as diagnostic of M.B.D. as some had thought. This article reminds us that the diagnosis of etiology is good medical practice. There are multiple causes for the behavior of hyperactivity. Symptom amelioration alone has little effect on the etiology of hyperactivity and may do harm to the child by not helping the underlying problem and by leading to unknown side effects in a developing organism.

(J. Tarnow, M.D.)

Hb Alc—An Indicator of Diabetic Control. Tze WJ *et al. J Pediatr* 93:13 (1978)

In a study of juvenile-onset diabetics, Hb Alc values were compared with other commonly used indicators of diabetic control. Hb Alc was higher in long-term and newly diagnosed diabetics than in normals or diabetics in the "honeymoon" phase. There was also an excellent correlation between Hb Alc and a single observer "evaluation" of diabetic control and good correlations with fasting blood glucose and 24-hour urinary glucose levels.

Comment: This study documents the association between the amount of glycosalated hemoglobin—Hb Alc (now a commercially available test) and other assessments of diabetic control. It appears that while the duration and degree of hyperglycemia is not the only determinant of subsequent vascular complications, it is a significant one. This test provides the diabetologist and the practitioner with a quantitative, retrospective assessment of the patient with diabetes.

Although other tools such as 24-hour fractional quantitative urine glucose (*Pediatrics* 53:257, 1974) and dextrostick/reflectometer self-monitoring of blood glucose (*Lancet* 1:729 and 731, 1978) are more useful for correcting difficulties in management, the Hb Alc test at least alerts us to the fact that the difficulties are present.

(S. Kleeman, M.D.)

Nutrient Deficiencies in Breast-fed Infant. Fomon SJ *et al. N Engl J Med* 299:355 (1978) (editorial)

Vitamin K deficiency is more common in breast-fed infants. Vitamin D may be marginal. Iron, although well absorbed, may not be in sufficient quantity. Fluoride is not adequate even if the mother is supplemented. Infants with problems may need supplementation (e.g., premature, vitamin dependency diseases). Poor nutrient intake in the mother (e.g. strict vegetarianism) may lead to infant deficiencies. Human milk is "unquestionably a superb food . . . however . . . (it) is neither a perfect nor a complete food."

Cows' Milk as a Cause of Infantile Colic in Breast-Fed Infants. Jakobsson I *et al. Lancet* 2:437 (1978)

In a noncontrolled study mothers of 19 infants with colic who were breast fed were put on a cows' milk, protein-free diet. Colic abated. Various challenges led to colic again and discontinuing led to improvement. Breast milk of mothers ingesting cows' milk contained cows' milk protein antigen which disappeared when the mother was off cows' milk.

Comment: Wow! The infant can get cows' milk protein from the mother's breast! Does this matter? This very preliminary report suggests that it might. At least it is food for thought and flight of ideas.

(R.H. Rapkin, M.D.)

Does That Child Really Have Cystic Fibrosis? Smalley CA *et al. Lancet* 2:415 (1978)

Sweat tests were falsely positive in 14 children. Most were tested at hospitals where sweat tests rarely are done. Attention to the detail of sweat collection is critical to an appropriate analysis.

Comment: False-positive sweat tests usually result from collecting too little sweat and the resultant evaporation leads to a falsely elevated concentration of electrolytes. Referral to a regional center always should be done to confirm the diagnosis of cystic fibrosis and to plan management. This problem disease can be cared for by the primary care practitioner under the guidance and direction of an expert in the disease.

(R.H. Rapkin, M.D.)

Late Effect of Nocturnal Mist Tent Therapy Related to the Severity of Airway Obstruction in Children with Cystic Fibrosis. Bureau MA *et al. Pediatr* 61:842 (1978)

In a controlled study two groups of children with C/F matched for severity were studied on and off mist tent therapy. No differences were found in the progression of airway obstruction. No benefit was found regardless of severity at onset of therapy.

Comment: Since mist therapy increases the risk of "waterbug" infection (Pseudomonas, Serratia, and others) and since it appears to be of little or no value in several studies, perhaps we need to rethink our treatment plans. Lower airway disease whether acute or chronic appears to be benefited rarely by mist and, with the exception of croup, its use should be questioned more frequently.

(R.H. Rapkin, M.D.)

Newborn Head Size and Neurological Status. Gross SJ *et al. Am J Dis Child* 132:753 (1978)

Head circumference at birth less than 10th percentile was a good predictor for poor growth and development at five years of age. There was a strong correlation with intellectual development.

Comment: Best to be appropriately grown for gestational age. Less good to be skinny but normal in length and head circumference. Worse is small and short for gestational age and even poorer prognostically is low head circumference. As all good pediatricians know all infants should be weighed carefully and measured at birth and their parameters plotted on a gestational age chart (after assessment of same by Dubowitz or other score). Prognosis then can be inferred and infants at higher risk than normal followed more carefully.

(R.H. Rapkin, M.D.)

The Use of Developmental Screening Tests by Primary Care Pediatricians. Smith RD *J Pediatr* 93:524 (1978)

Most pediatricians do not evaluate development by using any formal tests such as developmental screening tests. More recently trained pediatricians used formal tests.

Comment: It is well known that even experienced clinicians cannot accurately appraise intelligence without testing. That pediatricians are not doing this is a disturbing fact, if confirmed. Since most of what a pediatrician does is health maintenance, screening for treatable aberrations like mild mental retardation should be his stock and trade. Why not? The author points out that this might be an area where physician extenders could be used.

(R.H. Rapkin, M.D.)

THERAPEUTIC DRUG INFORMATION

This information is compiled by the Schwartz Inter-National Pharmaceutic and Therapeutic Drug Information Center of the Brooklyn College of Pharmacy, Long Island University.*

1. It has come to my attention that aspirin may have an effect on vitamin C requirements. Could you give more information on this possible interaction?

Vitamin C, an essential vitamin for man, is stored in the leukocytes and platelets.[1,2] The level of leukocyte ascorbic acid is a measurement of the tissue status of the vitamin when all metabolic demands are made. The physiological functions of vitamin C are not yet known, but it does act as a cofactor in enzymatic hydroxylation reactions.[3] The metabolic demand for the vitamin is increased under certain stressful situations including chronic disease states; eg., rheumatoid arthritis, the common cold, chronic and acute respiratory symptoms of allergic and infective etiology, and others.[1,2,4]

Loh *et al*[4] studied the interaction of aspirin and ascorbic acid on six healthy male volunteers. In was found, after a loading dose of 500mg of vitamin C, increasing doses of aspirin altered the plasma and leukocyte ascorbic acid concentrations. The plasma ascorbic acid levels rose while the leukocyte levels of the vitamin fell. This indicated impaired leukocyte absorption of the vitamin and a dose of 600mg of aspirin completely inhibited absorption of ascorbic acid into the leukoycytes.

Loh *et al*[1] in another study looked at the effects of aspirin on the metabolic availability of ascorbic acid. Results suggested that aspirin competes for ascorbic acid receptors in the leukocyte membrane and thus inhibits uptake of the vitamin.

Shaud and Cohen[2] examined the effect of aspirin ingestion on platelet and plasma levels of ascorbic acid in 48 normal subjects and 34 patients with confirmed rheumatoid arthritis. Significantly low platelet and plasma levels of ascorbic acid were found in those rheumatoid patients receiving high doses of aspirin who did not supplement their vitamin C intake.

In conclusion the interaction of aspirin and ascorbic acid may be of importance to the patient who is taking aspirin chronically. These patients probably should be advised to supplement their vitamin C intake.

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2. Do you have any information concerning the use of propranolol on a once-daily dosage schedule for hypertension?

Propranolol (Inderal®) is used in the management of hypertension either alone or in combination with other antihypertensive agents. The usual effective dose range of propranolol for hypertension is 160 to 480mg per day, and it is recommended to be administered in divided doses.[1] There seems to be a discrepancy between the biological effect(s) and serum concentration of propranolol.[2,3] The biological activity of propranolol persists for eleven hours, as determined by inhibition of tachycardia induced by isoproterenol (Isuprel®), while the pharmacological half-life appears to be two to three hours. Since a multiple daily-dose schedule can sometimes result in poor patient compliance, attempts have been made in the last few years to reduce the number of daily doses of propranolol in the management of hypertension.

Westerlund and Hansson[2] studied the effect of propranolol administered once daily in 19 patients with moderate hypertension who previously were controlled with propranolol twice daily. The patients were instructed to take their total daily dose of the drug at one time in the morning and blood pressure was measured 24 hours later each day. After 15 weeks on the new treatment regimen, there was no significant difference in the mean blood pressure between the two dosage regimens.

Wilson *et al*[4] reported a study designed to investigate whether once-daily administration of propranolol controls the blood pressure as effectively as divided daily doses. A group of eleven patients were selected whose blood pressure had been controlled satisfactorily with propranolol adminis-

*The Center serves as a source of intelligence on therapeutic and pharmaceutic information not readily available to physicians, at no charge to them, and provides this information with minimal time involvement. It is staffed by trained pharmacists; Jack M. Rosenberg, Pharm.D., Associate Professor and Chairman, Division of Clinical Pharmacy, Brooklyn College of Pharmacy, is Director and Walter Modell, M.D., Emeritus Professor of Pharmacology at Cornell University Medical College, is pharmacologist consultant. The service is available Monday through Friday from 9 a.m. to 5 p.m.—telephone (212) 622-8989 or 330-2735. Responses to these questions were prepared by J. M. Rosenberg, M.S.; Pharm.D.; T. H. Chin, Pharm.D.; R. J. Fuentes, R. Ph.; D. L. Menkes, R. Ph.; S. H. Kaplan; Mollie Fisch.

tered three times daily and a diuretic. The patients were given the exact same treatment except that propranolol was administered all at one time daily. Over a period of three months, the mean blood pressure of patients on the once-daily therapy was not different compared to the divided-dosage regimen. No observation of an increase in adverse effects was reported for the new regimen.

In conclusion, the current recommended dosage schedule for propranolol in the management of hypertension is to administer the drug in two or three divided doses per day. It appears from the studies reviewed that single daily dose propranolol therapy was utilized successfully in treating hypertension. However it has not been established that this regimen is as effective as giving the drug in divided daily doses because the population tested was too small to draw a valid conclusion. Further long-term controlled clinical studies are required to establish the efficacy of a single daily dosage schedule of propranolol in hypertension.

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3. What is the current thinking on the value of cranberry juice in the treatment of urinary problems?

Cranberry juice is reputed to be useful as an adjunct in the treatment of chronic urinary tract infections, reduction of urine ammoniacal malodors, and possibly useful in controling calcium urolithiasis. It is a commerically variable product and its constituents have not been linked to a specific pharmacological effect in man.[3]

Although not proved, benzoic and quinic acid are two organic acids in cranberry juice that may play a role in urinary bacteriostasis and reduction of ammoniacal odors. Both acids serve as a chemical precursor to hippuric acid. While hippuric acid has been shown to be bacteriostatic to common urinary tract pathogens at a concentration of 0.02 to 0.04M at pH 5.0, this activity decreases considerably at a slightly higher pH[2]. It is important to note that at maximally tolerable daily amounts of cranberry juice (0.6 to 4.0 liters), the acidification of urine is often negligible and only occasionally does the hippuric acid level reach the required level that would be bacteriostatic at pH 5.0. Whether there are other antibacterial substances in cranberry juice of clinical importance is unanswered to date.[3]

A study assisted by Ocean Spray Cranberries Inc., conducted on incontinent patients on a female ward, reported a subjective reduction in ammoniacal odors related to cranberry juice ingestion[1]. However, analysis of urine pH and air ammonia showed no statistical differences from controls.

Light *et al*[3] reported on the effect of cranberry juice in patients with renal calcium stones. Ingestion of the juice did not influence total excretion of calcium in urine, but it did decrease ionized calcium in patients with renal calculi. The authors indicated that any beneficial effect may result from slight acidifying properties of the juice and/or possibly a decrease in ionized calcium in patients with renal calculi.

In conclusion, there is a paucity of objective data to support the use of cranberry juice for therapeutic purposes. Its urine-acidifying properties are unreliable.

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4. Is there an interaction between dexamethasone and phenytoin during concurrent administration?

Reports that higher than usual doses of dexamethasone (Decadron®) were required for patients on chronic phenytoin (Dilantin®) undergoing a plasma cortisol-dexamethasone suppression test suggested the possibility of a drug-drug interaction.[1,2]

Subsequently, studies in rats and humans indicated that phenytoin may accelerate the hepatic conjugation and biliary excretion of dexamethasone.[2] Human studies also have suggested that phenytoin may increase the metabolism of dexamethasone by enhancing its conversion to unconjugated metabolites of greater polarity.[3]

Ranauden[4] cited 20 patients with supratentorial tumors who required greater than normally prescribed doses of dexamethasone to control cerebral edema; 18 of the 20 patients had been maintained on phenytoin. Vincent[5] also reported on two patients with glioblastomas who had improved symptomatology on dexamethasone and phenytoin for six to eight weeks until regressions occurred. Instead of increasing the dose of dexamethasone, which would be normally indicated, the substitution of equipotent doses of methylprednisolone (Medrol®) resulted in prompt clinical improvement, thus indicating a possible interaction between dexamethasone and phenytoin. Previously, methylprednisolone and its succinate salt (Solu-Medrol®) had been demonstrated to be minimally affected by concurrent phenytoin administration.[5]

It is difficult to evaluate quantitatively the possible interaction between dexamethasone and phenytoin. Therefore, when these two drugs are utilized concurrently, the possibility of a diminished dexamethasone response should be considered. Methylprednisolone apparently is unaffected by this interaction.

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5. Does quinidine alter serum digoxin levels?

Reports from two independent groups indicated that the combination digoxin and quinidine resulted in elevated serum digoxin levels.

A study was conducted in Sweden on 12 patients maintained on digoxin for atrial fibrillation or flutter, whose therapeutic regimen was supplemented with quinidine bisulfate, 750 mg orally twice daily for three to five days.[1] All 12 patients experienced a rise in plasma digoxin concentra-

tions during concomitant quinidine therapy, and the mean digoxin level rose from 0.85 ng/ml to 1.6 ng/ml. The authors suggested that, while the mechanism of interaction was not yet defined, quinidine may affect the binding of digoxin in the tissues.

In another study, serum digoxin levels were observed in 27 patients on steady state doses of digoxin, who were not taking any other antiarrhythmic drugs, before and during quinidine therapy.[2] In 25 patients the serum digoxin level rose signficantly during quinidine therapy from a mean level of 1.4 ng/ml to 3.2 ng/ml in spite of a reduction in the mean dose of digoxin from 0.31 mg to 0.25 mg. New anorexia, nausea, or vomiting developed in 16 patients on combination therapy, and disappeared in the ten patients whose digoxin dose was lowered or held. Four patients with prior ventricular arrhythmias experienced a worsened arrhythmia within five days after quinidine was begun. Of 13 patients without prior ventricular premature beats, three developed this symptom in the same time period. One of those patients manifested ventricular fibrillation, one had asystole, and there was one sudden death. In four patients, it was established that the elevation in serum digoxin concentration occurred within 24 hours of quinidine therapy, and a higher level was sustained for several days. The authors suggest a kinetic interaction between quinidine and digoxin.

In conclusion, two studies suggest a significant increase in serum digoxin levels after instituting quinidine therapy. As a precaution it would be advisable to monitor the serum digoxin levels carefully when quinidine is added to the therapeutic regimen.

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6. There have been many inquiries about the availability of "salt-poor" albumin. Is this product still available?

"Salt-poor" albumin is still available, but the term "salt-poor" has been deleted from the name. The first normal serum albumin product, made about 40 years ago, was a 25 percent hypotonic, acidic solution in water. This was subsequently replaced with preparations adjusted to a neutral pH and a sodium concentration of 150 mEq/Liter.[1,2] The formula was not suitable for the possibly adverse storage conditions associated with military use, and they adopted another preparation formulated with high sodium concentration. By 1945, with the use of other stabilizers, the sodium concentration could be lowered to the range of 130 to 160 mEq/Liter, and this new solution was called "salt-poor" to distinguish it from the solution with the higher sodium content. Soon after the introduction of the "salt-poor" solution, the high sodium product was discontinued making the term "salt-poor" unnecessary; but it persisted for more than 30 years. New FDA regulations require that all plasma products, including albumin, have a sodium content range of 130 to 160 mEq/Liter.[3]

Products which already have been manufactured will not have the name "salt-poor" deleted from their label, but newly manufactured products will. Therefore, until stocks of older material have been consumed, one may encounter products bearing the term "salt-poor" but the products all have sodium content in the range of 130 mEq/Liter-160 mEq/Liter.[1]

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COMMENTARY

How Dangerous Is Marihuana?

Dr. Elmar Lutz quotes the late renowned anthropologist Margaret Mead as saying that it is "no more dangerous than alcohol or cigarettes and should be legalized for anyone over the age of sixteen" and goes on in his article (pages 253-259, this issue) to present brief reports of several cases of psychosis following marihuana use and a lengthy review of the literature. He concludes that it is "profoundly destructive to man and society" . . . "a dangerous drug, and legalization therefore may not be in the public interest." Dr. Mead's position is to the far left in this controversy on the harmfulness or harmlessness of marihuana. Dr. Lutz is certainly to the right of center but there are many whose opinions are more extreme. Extremists tend to present their feelings, rather than scientific findings; they may present preliminary findings, not yet confirmed by independent research, as if they were more; they may extrapolate too far from *in vitro* or animal experiments to man, and from one culture to another, without making clear the limitations of this approach. Few researchers are so modest as to underestimate the importance of their work; most exaggerate the meaning of their findings, especially when facing a television camera. Thus reports in the media have asserted that marihuana acts like DDT in the brain, that it is as teratogenic as thalidomide, that it causes brain atrophy, that cannabis smoke is more carcinogenic than tobacco smoke. The opposition asserts that marihuana is safe, that its greatest danger is the risk of arrest and incarceration; that as a safer substitute for alcohol it may be used to cure alcoholism, as well as to treat glaucoma, epilepsy, dysmenorrhea, and other disorders. Perhaps the truth is somewhere between these extremes.

ALCOHOL AND TOBACCO

There is no question about marihuana's lack of lethality as compared with alcohol and tobacco. Alcohol is the most used and most abused psychoactive substance. Used by about 70 percent of the United States population, it is probably abused by more than nine million people. Six percent of high school seniors consume alcohol daily. More than 200,000 alcohol-related deaths are reported yearly, eight percent of all deaths in the United States.

Tobacco is the second most used substance. Fifty-five million Americans smoke cigarettes daily. More than 300,000 die prematurely each year from smoking-related illnesses. By age 12, one out of five youngsters smokes; about 22 percent of youths and 40 percent of adults are regular smokers.

MARIHUANA

Marihuana is the most used illegal drug. It is estimated that 24,000,000 Americans have tried it at least once and that 16,000,000 have used it in the past month. Four million of these are between 18 and 25 years old. Ten percent of high school seniors are daily users. One death has been reported as due to marihuana overdose but even this is disputed, as no cannabis was found in the body tissues although a cannabis metabolite was found in the urine.[1] Compared to alcohol and tobacco the risk of death from marihuana is extremely low.

Marihuana is used because of its psychological effects. It almost always produces a pleasant, positive emotional state. Visual imagery may become more vivid; the user becomes aware of subtle qualities of sound rhythms; touch, taste, and smell sensations are enhanced. Time perception is characteristically changed; time may slow down or stop. In low doses it may potentiate social interaction but in high doses the effects on the mind may isolate the user from the group. Attacks of hilarity without due provocation may occur. Higher doses may lead to transient confusion, drowsiness, and impairment in short-term memory. Extraneous non-pharmacological factors play a part in the psychological effects of marihuana; such "placebo" factors include the social setting, the presence of external stimuli such as music or noise, the subject's experience and expectations, and of course, his personality. The only consistent physiological symptoms are conjunctival injection and tachycardia.

This much is relatively non-controversial. Generally both advocates and opponents agree that one should not operate a motor vehicle while under the influence of marihuana. The dangers of this are quite obvious. The controversy about the dangers of marihuana centers on the nature and frequency of its adverse effects and their propensity for causing physical, mental, or moral disability.

As cannabis preparations usually are smoked, one might expect to find reports of toxic effects on the respiratory tract. High frequencies of chronic bronchitis have been reported in heavy long-term users from Eastern populations, where mixtures of cannabis and tobacco are smoked. A study frequently cited investigated 22 American soldiers in West Germany who smoked huge quantities of hashish (100 Gm or more monthly) for 6 to 15 months.[2] They had many upper and lower respiratory complaints, including sinusitis, asthma, nasopharyngitis, and bronchitis. However, 21 of the 22 also smoked tobacco, and as hashish is about five times as potent as marihuana, the relevance of this study to chronic American marihuana use is not clear. One might conclude that the use of massive doses of cannabis, especially with tobacco, is unhealthy. A more recent study by Boulougouris[3,4] of Athens University, Greece, of 44 heavy long-term hashish users found 13 to have nondisabling bronchitis, while six of 38 non-user controls did also. This difference is not statistically significant but the fact that both groups were heavy tobacco smokers and that eight of the 44 hashish users had hepatomegaly due to alcoholism emphasizes the difficulty of doing carefully controlled field studies. Boulougouris considered that his data did not support the view that chronic cannabis use leads to physical deterioration.[3,4] However it seems more reasonable to expect as much respiratory irritation from cannabis preparations as from unfiltered tobacco, with heavy users suffering more than light users. Sidney Cohen,[5] former Director of the Division of Narcotic Addiction and Drug Use, National Institute of Mental Health, and a very astute observer of the drug scene, points out the need to follow large populations of users for long periods of time. He reminds us that it took more than 200 years to establish a link between tobacco and bronchogenic carcinoma although that disorder is not uncommon. Although we see few marihuana smokers with lung cancer, few tobacco smokers with lung cancer were recognized thirty years ago before we were alert to this relationship.

PSYCHOLOGICAL EFFECTS OF MARIHUANA

The situation is quite different with regard to adverse psychological effects, which are fairly frequent. "Bad trips" which resemble the acute panic reactions seen with other hallucinogens are common. Toxic-confusional states with disorientation, paranoid ideation, and sometimes hallucinations also occur and usually subside fairly quickly. Schizophreniform psychoses and depressions are seen less frequently. Some of these toxic psychologic reactions are due to inadvertent overdose; other predisposing factors include the user's inexperience and personality. Severe stress, preexisting psychopathology, and a family history of schizophrenia also have been reported. The psychotic episodes tend to be self-limited and of short duration if marihuana use is terminated. There is very limited information on how frequently toxic psychological reactions occur. In Vietnam five cases of acute psychosis associated with marihuana use per 45,000 troops per month were estimated to occur,[6] although the number of actual marihuana users was not known. The student health service at Yale reported eleven cases per 8,500 students for the 1968-69 year,[7] and Los Angeles County Hospital reported nine admissions resulting from marihuana use out of 700,000 admissions from 1961 to 1969 (five of these were admitted after intravenous use of marihuana).[8] These figures are virtually useless in estimating the risk of serious psychological reactions. Most researchers and students of cannabis use believe that it may precipitate a psychosis rather than cause one *de novo*. Its use is considered by most writers to be a contributing factor in the etiology of psychosis among drug users, and it certainly should be avoided by individuals who have had episodes of schizophrenia or other drug-induced psychosis.

Another controversial aspect of the psychology of marihuana users is the so-called "amotivational syndrome." Prolonged use of marihuana has been said to cause personality changes manifested by emotional lability, loss of interest and ambition, lack of self-esteem, poor personal hygiene, and pansexuality. Advocates of marihuana use argue that these are also attributes of many who voluntarily accept a new life style, reject traditional values and roles, and "drop out." Opponents of marihuana assert that the use of the drug is responsible for the changes in personality, and it is reasonable to accept that heavy users who are chronically and constantly intoxicated cannot be expected to show conventional levels of motivation, apart from that required to obtain enough of the drug. One recent study from the Institute for Research in Social Behavior, University of California at Berkeley, studied a population of undergraduates where cannabis use was statistically normal rather than deviant. Glen D. Mellinger and his associates[9] studied 960 entering University of California freshmen in the fall of 1970 and resurveyed 834 of them in the spring of 1973. Of those who had never used marihuana, three percent had dropped out of school compared with six percent of those who used marihuana only and 14 percent who used a combination of psychoactive drugs. Dr. Mellinger's detailed analysis of his data led him to conclude that marihuana use is less a cause than an effect of such underlying psychosocial factors as the student's initial motivation for higher education, relationship with parents, and (among the multi-drug users) parental college degree.

Other claimed effects or adverse effects of marihuana, such as enhancement of sexual potency, impairment of sexual potency, impairment of fertility, cerebral atrophy, chronic organic brain syndrome, chromosome breakage, convulsant action, anticonvulsant action, antihypertensive therapeutic effects, appetite stimulating therapeutic effects, potential for treating withdrawal effects of alcohol, barbiturates or opiates —all these and many others—are inadequately documented at the present time and their potential relevance is debatable.

The past few years have seen the isolation of the active natural cannabinoids and their synthesis in quantity. The National Institute of Mental Health has made generous supplies available for research. It will be some time before laboratory research catches up with the results of the crude natural experiments of the workers in the field, who have at least 4000 years of experience with cannabis. Meanwhile our efforts should be aimed at educating the citizens and legislators about known medical data with perhaps some estimates of how likely the data are to be true. Drug education in the schools has proved a dismal and expensive failure, with neither the traditional "scare approach" or the more recent "information-transmission approach" proving useful; in fact, both have been accused of stimulating rather than deterring drug use. Drug education of legislators has led to the decriminalization of possession of small amounts of marihuana in many states, with no obvious ill effects thus far. No country has seen fit to legalize marihuana use and there is no pressing need for this here.

Is marihuana as dangerous as alcohol and tobacco? More dangerous? Less dangerous? From the point of view of mortality, it is much less dangerous. From the point of morbidity, the answer must be "It depends. . . . " It depends on the personality of the user, on the potency of the marihuana, on the frequency of its use and duration of its use. Occasional use by mentally healthy adults so far has not been proved to be more dangerous than an occasional cocktail; heavy use is probably as conducive to ill health as heavy use of alcohol and tobacco, and it is likely that similar personality features predispose to psychological dependence on these and other drugs. Marihuana use cannot be suppressed and certainly should not be encouraged. Until harder evidence is available the claims of both ardent supporters and zealous opponents of the drug should be viewed with proper scientific scepticism.

A. Arthur Sugerman, M.D.

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Report of the Nominating Committee

Offices To Be Filled by Election—1979 Annual Meeting

Frank R. Begen, M.D., Chairman

Office	Term	Nominee and County
President-Elect	1 year	George L. Benz, M.D., Essex
1st Vice-President	1 year	Augustus L. Baker, Jr., M.D., Morris
2nd Vice-President	1 year	Armando F. Goracci, M.D., Gloucester
Trustees:		
1st District	3 years	Myles C. Morrison, Jr., M.D., Morris
4th District	3 years	Meyer L. Abrams, M.D., Burlington
4th District	3 years	Anthony P. DeSpirito, M.D., Momouth
5th District	3 years	*No Nominee
Judicial Councilor:		
3rd District	3 years	Albert F. Moriconi, M.D., Mercer
AMA Delegates:	2 years	William J. D'Elia, M.D., Monmouth
	2 years	James S. Todd, M.D., Bergen
**AMA Alternate Delegates:	2 years	Howard D. Slobodien, M.D., Middlesex
	2 years	Edward A. Schauer, M.D., Monmouth
	2 years	Frank Y. Watson, M.D., Essex
	2 years	James H. Spillane, M.D., Warren
New York:		
Delegate	1 year	Albert F. Moriconi, M.D., Mercer
Alternate	1 year	F. Sterling Brown, M.D., Atlantic
Connecticut:		
Delegate	1 year	Edward M. Coe, M.D., Union
Alternate	1 year	Gastone A. Milano, M.D., Atlantic
Administrative Councils:		
Legislation:		
2nd District	3 years	John J. Crosby, Jr., M.D., Hudson
3rd District	3 years	Leon A. Fraser, M.D., Mercer
Medical Services:		
2nd District	3 years	John R. Doyle, M.D., Bergen
3rd District	3 years	William E. Ryan, M.D., Mercer
Mental Health:		
4th District	3 years	+ No Nominee
5th District	3 years	+ No Nominee
Public Health:		
2nd District	3 years	Albert Ehrlich, M.D., Hudson
3rd District	3 years	Thomas E. Desmond, M.D., Middlesex
Public Relations:		
3rd District	3 years	Milton R. Bronstein, M.D., Middlesex
6th Member	3 years	Edwin W. Messey, M.D., Burlington
Standing Committees:		
Annual Meeting	3 years	Joseph P. Cillo, M.D., Union
Auxiliary Advisory	3 years	Frank R. Romano, M.D., Union
Finance and Budget	3 years	Palma E. Formica, M.D., Middlesex
Medical Defense and Insurance	3 years	Michael J. Doyle, M.D., Monmouth
Medical Education	3 years	William Pomerantz, M.D., Morris
Publication	3 years	Paul J. Hirsch, M.D., Somerset

*Incumbent nominated for second Vice-President

**The Committee will reconvene on March 28 to review the procedures taken in the process of nominating the AMA Alternate Delegates

+Biographical data not supplied, therefore no nominations were processed

DOCTORS' NOTEBOOK

Tumor Clinic/Tumor Board in Community Hospital*

In 1932 the Medical Staff of the Paterson General Hospital, mindful of the problems in treating cancer, requested the administration of the New York Memorial Hospital to establish a Tumor Clinic. Doctor George T. Pack, an outstanding and upcoming cancer specialist and chief of two separate cancer services at Memorial Hospital, was recommended. After meeting with the chiefs of services at the Paterson General Hospital, the groundwork was established and, with the generous financial help of the Lendrim family of Paterson, the Josephine Lendrim Tumor Clinic was organized. This was the first such clinic in New Jersey and one of the first in the United States. During the next thirty years, Doctor Pack gave unstintingly of his time and energy to the clinic every Saturday, working as long as 12 to 14 hours. After seeing as many as 75 to 100 patients, he would perform surgery beginning as late as 4 or 5 p.m.

As months became years, and as more and more patients came from outside the Paterson area to be treated, the reputation of the clinic spread throughout northern New Jersey. The success of the clinic became a tribute to the vision of those physicians who foresaw its need. It then became a seat of learning for the medical staff of the Paterson General Hospital. Many physicians attended the Saturday sessions, and became skilled in the treatment of cancer.

In addition to operating its own tumor clinic with a staff of over 100, and performing complicated and pioneer surgery (he performed the first liver resection in Memorial Hospital), Doctor Pack traveled widely. He lectured, taught, and served on many international organizations as president and vice-president. He contributed more than 450 scholarly papers, edited and co-edited several encyclopedias and many books. He was awarded seven honorary degrees and his influence and reputation became international in scope.

THE MAN

Doctor Pack was a lifetime student and teacher. He taught a great number of American physicians and helped to train many Latin American oncologists. His master was the scientific fact. Always striving to improve his own ability and knowing the value of advances in medical and surgical procedures, his self-evaluation and self-determination became his hallmarks of success. He was a great stimulus to sharpen the skills and improve the knowledge of, not only those who worked with him, but the entire staff of the Paterson General Hospital. The Medical Department became an integral part of the clinic, and was one of the first examples of a coordinated program with the various services cooperating for the benefit of the patient. He was a great contributor to continuing medical education. Doctor Pack died in his 70th year on January 23, 1969.

THE CLINIC—THE BOARD

The Tumor Clinic, as originally conceived by Doctor Pack, was a facility for the diagnosis and treatment by various modalities of all types of neo-plastic disease. The Tumor Board, however, has the added thrust of improved treatment and education. The keystone of a successful program is the contribution to better patient care by fulfilling educational and consultive functions. In the larger institution, the focus is on specific cancer cases that represent difficult management problems. In the Tumor Board of the small hospital, such as Paterson General, it is possible to review all cancer cases, including a follow-up. Meetings must be held often and at regular intervals. Members of the Board (made up of the staff physicians) become a forum for the exchange of ideas on treatment, diagnosis and management, rather than the expression of only specialist expertise. Cases are reviewed, and each member interprets information in light of his knowledge.

Precedent for case review is established in every hospital by the Joint Commission on Accreditation of Hospitals. This involves the Tissue, Utilization Review, and Cancer committees. This review can pinpoint specific areas where educational programs may be needed. Informal discussion of active cases stimulates the implementation of new knowledge. A physician whose case is being reviewed should attend the Tumor Board meeting. His case can be abstracted and reviewed by the Chairman. Comments and questions are solicited from those in attendance and management alternatives suggested.

Errors in judgment can be minimized or prevented by this review. A physician who does not recognize that he may be ignorant of certain diagnostic points and sophisticated treatment techniques can become informed. Consultation with experts in the field can be obtained or the patient can be referred to a special center.

The effectiveness of the Tumor Board Conference as a multi-disciplinary medium with all allied health para-professionals will be to the patient's advantage. If the Board functions properly, it can be a valuable educational instrument available to all members of the hospital staff and the foundation of a successful cancer program.

SUMMARY

Forty-five years ago, the first cancer clinic in New Jersey was established at the Paterson General Hospital by Dr. George T. Pack. His dedication to cancer cure, research, and teaching was the forerunner of continuing medical education and presaged the changes to come. Tumor boards, which are replacing tumor clinics, enhance the physician's knowledge and improve patient care.

*Based on a paper prepared by John A. Ianacone, M.D., Emeritus, Department of Surgery, Greater Paterson General Hospital, and read before the George T. Pack Memorial Cancer Symposium, Wayne, May 20, 1978.

Treatment Schedules for Gonococcal Infections—1978*†

UNCOMPLICATED GONOCOCCAL INFECTIONS IN MEN AND WOMEN

Drug Regimens of Choice

(1) Aqueous procaine penicillin G (APPG) 4.8 million units injected intramuscularly at two sites, with 1.0 g of probenecid by mouth.

(2) Tetracycline hydrochloride** 0.5 g by mouth four times a day for five days (total dosage 10.0 g). Other tetracyclines are not more effective than tetracycline hydrochloride. All tetracyclines are ineffective as a single-dose therapy.

(3) Ampicillin 3.5 g, or amoxicillin 3.0 g, either with one g probenecid by mouth. Evidence shows that these regimens are slightly less effective than the other recommended regimens.

(4) Patients who are allergic to the penicillins or probenecid should be treated with oral tetracycline as above. Patients who cannot tolerate tetracycline may be treated with spectinomycin hydrochloride 2.0 g in one intramuscular injection.

Special Considerations

—Single-dose treatment is preferred in patients who are unlikely to complete the multiple-dose tetracycline regimen.

—The APPG regimen is preferred in men with anorectal infection.

—Pharyngeal infection is difficult to treat; high failure rates have been reported with ampicillin and spectinomycin.

—Tetracycline treatment results in fewer cases of postgonococcal urethritis in men.

—Tetracycline may eliminate coexisting chlamydial infections in men and women.

—Patients with incubating syphilis (seronegative, without clinical signs of syphilis) are likely to be cured by all the above regimens except spectinomycin. All patients should have a serologic test for syphilis at the time of diagnosis.

—Patients with gonorrhea who also have syphilis or are established contacts to syphilis should be given additional treatment appropriate to the stage of syphilis.

Treatment of Sexual Partners—Men and women exposed to gonorrhea should be examined, cultured, and treated at once with one of the regimens above.

Follow-up—Follow-up cultures should be obtained from the infected site(s) three to seven days after completion of treatment. Cultures should be obtained from the anal canal of all women who have been treated for gonorrhea.

Treatment Failures—The patient who fails therapy with penicillin, ampicillin, amoxicillin, or tetracycline should be treated with 2.0 g of spectinomycin intramuscularly.

Most recurrent infections after treatment with the recommended schedules are due to *reinfection* and indicate a need for improved contact tracing and patient education. Since infection by penicillinase (β-lactamase)-producing *Neisseria gonorrhoeae* is a cause of treatment failure, post-treatment isolates should be tested for penicillinase production.

Not Recommended—Although long-acting forms of penicillin (such as benzathine penicillin G) are effective in syphilotherapy, they have no place in the treatment of gonorrhea. Oral penicillin preparations such as penicillin V are not recommended for the treatment of gonococcal infection.

PENICILLINASE-PRODUCING *NEISSERIA GONORRHOEAE* (PPNG)

Patients with uncomplicated PPNG infections and their sexual contacts should receive spectinomycin 2.0 g intramuscularly in a single injection. Because gonococci are very rarely resistant to spectinomycin and reinfection is the most common cause of treatment failure, patients with positive cultures after spectinomycin therapy should be retreated with the same dose.

A PPNG isolate that is resistant to spectinomycin may be treated with cefoxitin 2.0 g in a single intramuscular injection, with probenecid 1.0 g by mouth.

TREATMENT IN PREGNANCY

All pregnant women should have endocervical cultures for gonococci as an integral part of the prenatal care at the time of the first visit. A second culture late in the third trimester should be obtained from women at high risk for gonococcal infection.

Drug regimens of choice are APPG, ampicillin or amoxicillin, each with probenecid as described above.

Women who are allergic to penicillin or probenecid should be treated with spectinomycin.

Refer to the sections on acute salpingitis and disseminated gonococcal infections for the treatment of these conditions during pregnancy. Tetracycline should not be used in pregnant women because of potential toxic effects for the mother and fetus.

ACUTE SALPINGITIS (PELVIC INFLAMMATORY DISEASE)

There are no reliable clinical criteria on which to distinguish gonococcal from nongonococcal salpingitis. Endocervical cultures for *N. gonorrhoeae* are essential. Therapy should be initiated immediately.

(a) Hospitalization should be strongly considered in these situations:

1. Uncertain diagnosis, in which surgical emergencies such as appendicitis and ectopic pregnancy must be excluded.
2. Suspicion of pelvic abscess.
3. Severely ill patients.
4. Pregnancy.
5. Inability of the patient to follow or tolerate an outpatient regimen.
6. Failure to respond to outpatient therapy.

(b) Antimicrobial Agents

—Outpatients

(1) Tetracycline* 0.5 g taken orally four times a day for ten days. This regimen should not be used for pregnant patients.

(2) APPG 4.8 million units intramuscularly, ampicillin 3.5 g or amoxicillin 3.0 g each with probenecid 1.0 g. Either regimen is followed by ampicillin 0.5 g or amoxicillin 0.5 g orally four times a day for ten days.

—Hospitalized patients

(1) Aqueous crystalline penicillin G 20 million units given intravenously each day until improvement occurs, followed by ampicillin 0.5 g orally four time a day to complete ten days of therapy.

(2) Tetracycline* 0.25 g given intravenously four times a day until improvement occurs, followed by 0.5 g orally four times a day to complete ten days of therapy. This regimen should not be used for pregnant women. The dosage may have to be adjusted if renal function is depressed.

(3) Since optimal therapy for hospitalized patients has not been established, other antibiotics in addition to pencillin frequently are used.

*From the Center for Disease Control, US Public Health Service, DHEW, Atlanta, Georgia.

†Physicians are cautioned to use no less than the recommended dosages of antibiotics.

**Food and some dairy products interfere with absorption. Oral forms of tetracycline should be given one hour before or two hours after meals.

(c) Special Considerations
—Failure of the patient to improve on the recommended regimens does not indicate the need for stepwise additional antibiotics but requires clinical reassessment.
—The intrauterine device is a risk factor for the development of pelvic inflammatory disease. The effect of removing an intrauterine device on the response of acute salpingitis to antimicrobial therapy and on the risk of recurrent salpingitis is unknown.
—Adequate treatment of women with acute salpingitis must include examination and appropriate treatment of their sex partners because of their high prevalence of nonsymptomatic urethral infection. Failure to treat sex partners is a major cause of recurrent gonococcal salpingitis.
—Follow-up of patients with acute salpingitis is essential during and after treatment. All patients should be recultured for *N. gonorrhoeae* after treatment.

ACUTE EPIDIDYMITIS

Acute epididymitis can be caused by *N. gonorrhoeae, Chlamydia* or other organisms. If gonococci are demonstrated by Gram stain or culture of urethral secretions, treatment should be:

(1) APPG 4.8 million units, ampicillin 3.5 g or amoxicillin 3.0 g, each with probenecid 1.0 g. Either regimen is followed by ampicillin 0.5 g or amoxicillin 0.5 g orally four times a day for ten days.

(2) Tetracycline** 0.5 g orally four times a day for ten days.

(3) If gonococci are not demonstrated, the above tetracycline regimen should be used.

DISSEMINATED GONOCOCCAL INFECTION

(a) Equally effective treatment schedules in the arthritis-dermatitis syndrome include:

(1) Ampicillin 3.5 g or amoxicillin 3.0 g orally, each with probenecid 1.0 g, followed by ampicillin 0.5 g or amoxicillin 0.5 g four times a day orally for seven days.

(2) Tetracycline* 0.5 g orally four times a day for seven days. Tetracycline should not be used for complicated gonococcal infection in pregnant women.

(3) Spectinomycin 2.0 g intramuscularly twice a day for three days (treatment of choice for disseminated infections caused by PPNG).

(4) Erythromycin 0.5 g orally four times a day for seven days.

(5) Aqueous crystalline penicillin G 10 million units intravenously per day until improvement occurs, followed by ampicillin 0.5 g four times a day to complete seven days of antibiotic treatment.

(b) Special Considerations
—Hospitalization is indicated in patients who may be unreliable, have uncertain diagnosis, or have purulent joint effusions or other complications.
—Open drainage of joints other than the hip is not indicated.
—Intra-articular injection of antibiotics is unnecessary.

(c) **Meningitis and endocarditis** caused by the gonococcus require high-dose intravenous penicillin therapy. In penicillin-allergic patients with endocarditis, desensitization and administration of penicillin is indicated; chloramphenicol may be used in penicillin-allergic patients with meningitis.

GONOCOCCAL INFECTIONS IN PEDIATRIC PATIENTS

With gonococcal infections in children beyond the newborn period the possibility of sexual abuse must be considered. Genital, anal, and pharyngeal cultures should be obtained from all patients before antibiotic treatment. Appropriate cultures should be obtained from individuals who have had contact with the child.

PREVENTION OF GONOCOCCAL OPHTHALMIA

When required by State legislation or indicated by local epidemiologic considerations, effective and acceptable regimens for prophylaxis of neonatal gonococcal ophthalmia include:

(1) Ophthalmic ointment or drops containing tetracycline or erythromycin.

(2) One percent silver nitrate solution.

Special considerations
—Bacitracin is not recommended.
—The value of irrigation after application of silver nitrate is unknown.

MANAGEMENT OF INFANTS BORN TO MOTHERS WITH GONOCOCCAL INFECTION

The infant born to a mother with gonorrhea is at high risk of infection and requires treatment with a single intravenous or intramuscular injection of aqueous crystalline penicillin G 50,000 units to full-term infants or 20,000 units to low-birth-weight infants. Topical prophylaxis for neonatal ophthalmia is not adequate treatment. Clinical illness requires additional treatment.

NEONATAL DISEASE

(a) **Gonococcal Ophthalmia:** Patients should be hospitalized and isolated for 24 hours after initiation of treatment. Untreated gonococcal ophthalmia is highly contagious. Aqueous crystalline penicillin G 50,000 units/kg/day in two doses intravenously should be administered for seven days. Saline irrigation of the eyes should be performed as needed. Topical antibiotic preparations alone are not sufficient or required when appropriate systemic antibiotic therapy is given.

(b) **Complicated Infection:** Patients with arthritis and septicemia should be hospitalized and treated with aqueous crystalline penicillin G 75,000 to 100,000 units/kg/day intravenously in two or three divided doses for seven days. Meningitis should be treated with aqueous crystalline penicillin G 100,000 units/kg/day, divided into three or four intravenous doses, and continued for at least ten days.

CHILDHOOD DISEASE

Children who weigh 100 lbs. (45 kg) or more should receive adult regimens. Children who weigh less than 100 lbs. should be treated as follows:

(a) **Uncomplicated Disease**—Uncomplicated vulvovaginitis, urethritis, proctitis, or pharyngitis can be treated at one visit with:

(1) Amoxicillin 50 mg/kg orally with probenecid 25 mg/kg (maximum 1.0 g).

(2) Aqueous procaine penicillin G 100,000 units/kg intramuscularly plus probenecid 25 mg/kg (maximum 1.0 g).

Special Considerations
Topical and/or systemic estrogen therapy are of no benefit in vulvovaginitis.
—Long-acting penicillins, such as benzathine penicillin G, are not effective.
—All patients should have follow-up cultures and the source of infection should be identified, examined, and treated.

Gonococcal Ophthalmia—Ophthalmia in children is treated as in neonates but the dose of penicillin is increased to 100,000 units/kg/day intravenously.

Complicated Infections—Patients with peritonitis or arthritis require hospitalization and treatment with aqueous crystalline penicillin G, 100,000 units/kg/day intravenously for seven days. Aqueous crystalline penicillin G

250, 000 units/kg/day intravenously in six divided doses for at least ten days is recommended for meningitis.

Allergy to Penicillins—Children who are allergic to penicillins should be treated with spectinomycin 40 mg/kg intramuscularly. Children older than eight years may be treated with tetracycline 40 mg/kg/day orally in four divided doses for five days. For treatment of complicated disease, the alternative regimens recommended for adults may be used in appropriate pediatric dosages.

CMDNJ Notes*

Stanley S. Bergen, Jr., M.D.
President

A recent study undertaken by CMDNJ and the Advisory Graduate Medical Education Council reveals that more American-educated physicians than ever before are choosing New Jersey hospitals for their graduate medical training. Furthermore, the study shows that the largest number of graduate medical education positions are available in hospitals throughout the state and more New Jerseyans are filling those positions than previously.

As chairman of the advisory council, I particularly am gratified by the study's revelations because, if these trends continue, more graduates of United States medical schools actually will be practicing in the state. The purpose of the study, which involved 39 hospitals, was to help guide the council in planning graduate medical education programs.

Specifically, the study showed that 39 percent more United States medical school graduates chose New Jersey hospitals in 1977-78 over the previous school year, while 37 percent more American graduates of foreign medical schools made the same decision. Between the two school years, according to the report, there also has been an increase of 251 of these internships and residencies—from 1,776 to 2,027. Significant to us is the fact that of the 920 United States citizens holding these positions, 359 are native New Jerseyans and 258 are CMDNJ graduates.

This increase speaks well for the medical manpower needs of the state because, as I've noted before, studies show a high percentage of doctors settle in areas near their hospital training, particularly when medical school affiliated. As many as 80 percent of these young physicians can be expected to settle within 50 miles of where they are taking their residency training. Considering our physician needs in New Jersey, it is critical that the council concentrate on developing quality programs to continue to attract medical school graduates.

The Advisory Graduate Medical Education Council was created by law a little more than a year ago. As an advisory group to the Board of Higher Education, the council identifies areas of physician need and recommends funding to develop or expand appropriate hospital-based residency training programs. The council's initial thrust is in the area of primary care programs for physician-short areas, such as in southern New Jersey. Six other members of the Medical Society of New Jersey serve with me on the council. They are: Brewster Miller, M.D. (Somerset), Arthur Bernstein, M.D. (Essex), Enio Calluori, M.D. (Essex), Samuel J. Lloyd, M.D. (Mercer), Leo H. Siegel, M.D. (Essex), and Nelson Walker II, M.D. (Hunterdon).

Results of the council's study have an added significance, in view of the limitations now placed on foreign medical graduates coming into this country. These physicians formerly filled as many as 85 percent of the state's residency places; however, they are being curtailed sharply by federal legislation aimed at reducing United States dependence on foreign physicians. The percentage of foreign physicians holding residency positions in the state has dropped from a high of 85 percent in 1973, to 55 percent this year.

The report also points out that New Jersey's dependence on foreign medical graduates stems mainly from the fact that there was no medical school in New Jersey until the late 1950's with creation of the former Seton Hall University Medical School, which became one forerunner of CMDNJ. The College today has six schools, including three flourishing medical schools, and a network of affiliated hospitals that crisscrosses the state. Our growing reputation is underscored by the increasing number of doctors who are coming to our hospitals for their residency training.

The state's dependence on foreign medical graduates is highest, according to the study, in the specialities of pathology, anesthesiology, psychiatry, and radiology. That dependence is much lower for family practice residencies. The report further reveals that New Jersey is well ahead of federal requirements that called for a minimum of 35 percent of first-year residencies in primary care by 1978. New Jersey had 54 percent last year.

Since enactment of the new immigration law, New Jersey was one of a number of states facing the prospect of residency positions going unfilled. Creation of the advisory council, however, was one significant measure taken by the state to ensure that diverse graduate opportunities would be available to attract American medical school graduates.

The state's concern about the graduate medical education system, in particular its effect on the maldistribution of physicians in the state, dates back to 1972 when an ad hoc group was formed under the auspices of CMDNJ to study the situation. Three years later, that council's recommendation for the formation of a coordinated, statewide, and state-supported system was proposed through legislation. After three years of testimony, and some amending, it was signed by Governor Byrne last winter.

The original ad hoc council consisted of the leaders of 16 major health and education agencies in the state. The Chancellor of Higher Education, the Commissioner of Health, and the Commissioner of Human Services, who were members of the ad hoc council, serve as ex-officio members of the new advisory council.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Office of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ALLERGY—Yalamanchi K. Rao, M.D. 354 Henry Street. Apt. #2, Brooklyn, NY 11201. Nagpur Medical College (India) 1968. Board eligible. Group, partnership, solo, institutional, research. Available July 1979.

Richard W. Huss, M.D. 555 Graham Road, Fort Sam Houston, TX 78234. CMDNJ 1971. Subspecialty, internal medicine. Board certified (IM). Group or solo. Available September 1979.

Andrew W. Green, M.D., 1 Raintree Island, Apt. 6, Tonawanda, NY 14150. St. Louis University 1974. Subspecialty, internal medicine. Board certified (IM). Solo or group. Available July 1979.

ANESTHESIOLOGY—Jitendra G. Patel, M.D., 133-52 Avery Avenue, Flushing, New York 11355. M. P. Shah (India) 1973. Board eligible. Any type practice. Available July 1979.

Rolycito A. Chan, M.D., 2121 S. Wolf Road, Hillside, IL 60162. Cebu (Philippines) 1974. Board eligible. Group, partnership, solo. Available July 1979.

Ajit V. Pai, M.D., 8136 Braeburn Terrace, Indianapolis, IN 46219. Topiwala National (India) 1973. Board eligible. Group or partnership. Available September 1979.

Mohammed A. Mohiuddin, M.D., 104 West Franklin Street, Apt. 908, Richmond, VA 23220. Osmania Medical College (India) 1971. Board eligible. Group or partnership. Available July 1979.

Kie C. Ohn, M.D., 520 Second Avenue, Apt. 11-F, New York, NY 10016 Seoul National (Korea) 1971. Subspecialty, family practice. Board eligible. Group, partnership, or solo. Available July 1979.

Jitrendra G. Patel, M.D., 133-52 Avery Avenue, Flushing, NY 11355. Jamnagar-Guj (India) 1973. Board eligible. Academic, research, or administrative post. Available July 1979.

Vinod Malhotra, M.D., 40 Prospect Avenue, Apt. 63, Hackensack 07601. University of Delhi (India) 1972. Board eligible. Group, partnership, or solo. Available July 1979.

Hyun Hwang, M.D., 435 East 70th Street, Apt 7-F, New York, NY 10021. Yonsei University (Korea) 1964. Board eligible. Group, partnership, solo, or industrial. Available July 1979.

Subbarao Narepalem, M.D., 2947 Northview Drive, Youngstown, OH 44504. Andhra Medical College (India). Board eligible. Group, partnership, fee for service. Available July 1979.

CARDIOLOGY—Masoud Javed, M.D., Deborah Heart and Lung Center, Browns Mills 08015. Liaquat Medical College (Pakistan). Subspeciality, internal medicine. Any type practice. Available July 1979.

CARDIOVASCULAR DISEASES—Il Young Kim, M.D., 7044 Glen Meadow Lane, Cincinnati, OH 45237. Seoul National University (Korea) 1971. Subspecialty, internal medicine. Board certified (IM). Group, partnership, institutional. Available July 1979.

Stephen M. Barolsky, M.D., 6347 Caton Street, Pittsburgh, PA 15217. SUNY—Downstate 1973. Subspecialty, internal medicine. Board certified (IM). Group, partnership, institutional, research. Available July 1979.

Rajinder K. Shalla, M.D., 353 East 17th Street, Apt. 8-F, New York, NY 10003. All-India Institute (India) 1968. Subspecialty, internal medicine. Board eligible (IM). Group, institutional. Available July 1979.

Vrajesh M. Parikh, M.D., 2520 South King Drive, Chicago, IL 60616. B.J. Medical College (India) 1970. Subspecialty, internal medicine. Board eligible (IM). Group, partnership, research. Available July 1979.

Shashibalao P. Sehgal, M.D., Rockefeller University, 1230 York Avenue, New York, NY 10021. Seth G.S. Medical College (India) 1973. Subspecialty, internal medicine. Board certified (IM). Group, partnership, public health, industrial, institutional. Available July 1979.

DERMATOLOGY—Fred V. Kemp, M.D., 114 East Bailey Road, Naperville, IL 60540. St. Louis University 1973. Group, partnership, or solo. Available July 1980.

EMERGENCY MEDICINE—Nicanor C. Lopez, M.D., 1432 Arch Street, Apt. 8202, Norristown, PA 19401. Far Eastern University (Philippines) 1967. Subspecialty, general practice. Group, partnership, emergency room. Available July 1979.

FAMILY PRACTICE—Timothy B. Eckel, M.D., 1410 Bloom Road, Danville, PA 17821. Temple 1976. Board eligible. Group or partnership. Available July 1979.

Rosario A. Bascon, M.D., 15 St. Andrews Place, Apt. 2-A, Yonkers, NY 10705. Cebu (Philippines) 1967. Board certified. Solo, partnership, group. Available.

GASTROENTEROLOGY—Alan M. Nelson, M.D., 41B Monroe Street, Milford, CT 06460. Georgetown 1974. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

Robert S. Goldblatt, M.D., 150 Division Street, Derby, CT 06418. George Washington University 1974. Subspecialty, internal medicine. Board eligible (IM). Group or partnership. Available July 1979.

Jon David Green, M.D., 509 Fairview Avenue, Apt. C-1, Orange 07050. CMDNJ 1974. Subspecialty, internal medicine. Board certified (IM). Group, partnership, solo. Available July 1979.

Hua-Lung Lin, M.D., 911 West Apollo Avenue, Tempe, AZ 85283. National Taiwan University 1972. Subspecialty, internal medicine. Board eligible (IM). Group, partnership, solo. Available July 1979.

Myung Woong Kim, M.D., 6582 Poplar Woods, Circle S., Germantown, TN 38138. Yonsei University (Korea) 1968. Subspecialty, gastroenterology. Board certified (IM). Group, partnership, solo. Available July 1979.

Vithal Kusuma, M.D., 1227 S. Harlem Street, Berwyn, IL 60402. Osmania (India) 1969. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

GENERAL PRACTICE—Dharamsi D. Shah, M.D., 22-4B Passaic Avenue, Nutley 07110. Grant Medical College (India) 1974. Subspecialty, psychiatry. Board eligible (psych.). Group, partnership, solo. Available July 1979.

James W. Warren, M.D., Ainsworth Health Clinic, Fort Hamilton, Brooklyn, NY 11252. Meharry 1973. Group, partnership, rural sponsorship, emergency room. Available.

HEMATOLOGY—Ming-Swang Lok, M.D., 163 Oxford Avenue, Buffalo, NY 14209. National Defense Medical Center (Taiwan) 1970. Subspecialty, internal medicine. Board certified (IM). Research or institutional. Available July 1979.

Subhash C. Proothi, M.D., 86-D Village Green North, East Providence, RI 02915. Rohtak Medical College (India) 1970. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

INFECTIOUS DISEASES—James E. Milder, M.D., 870 Johnsdale Drive, Lexington, KY 40505. Temple 1974. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

INTERNAL MEDICINE—Franciska Katona, M.D., Village Lane Apts., Oak-2, Abington, PA 19001. Temple 1976. Board eligible. Industrial, occupational, research, clinic, preventive medicine. Available July 1979.

Tsue-Pin Lin, M.D., 1945 Corlies Avenue, Neptune 07753. Kaohsiung (Taiwan) 1971. Subspecialty, general practice. Board eligible. Group, partnership, or solo. Available July 1979.

B. Madhusudhan Reddy, M.D., 69 Gold Street, Apt. 12-C, New York, NY 10038. Osmania Medical College (India) 1974. Board eligible. Group, partnership, institutional. Availavle July 1979.

Indu B. Patel, M.D., 9668 Dundawan Road, Baltimore, MD 21236. B.J. Medical College (India) 1971. Board eligible. Group, partnership, institutional. Available July 1979.

Rangachari Srinivasan, M.D., 1325 Pennsylvania Avenue, Apt. 15-D, Brooklyn, NY 11239. Stanley Medical College (India) 1970. Subspecialty, neoplastic diseases. Board certified. Group, partnership, solo. Available July 1979.

Mohamed Ziauddin, M.D., VA Center, Martinsburg, WV 25401. Madurai Medical College (India) 1969. Board eligible. Group, partnership. Available July 1979.

Robert I. Chapnick, M.D., 98-01 67th Avenue, Apt. 12-U, Forest Hills, NY 11374. SUNY—Downstate 1976. Subspecialty, emergency medicine. Board eligible. Group or partnership. Available August 1979.

Bui Ngoc Diep, M.D., 417 NE 13th Street, Oklahoma City, OK 73104. Faculty of Medicine (Vietnam) 1971. Subspecialty, neoplastic diseases. Board eligible. Group, partnership, institutional. Available July 1979.

Imdad H. Butt, M.D., Apt. 5B-1 Franklyn Manor 114 Franklyn Street, Morristown 07960. King Edward (Pakistan) 1970. Subspecialty, general practice. Board eligible (pediatrics). Partnership or solo. Available July 1979.

John S. Smiarowski, M.D., U.S. Naval Hospital, 7500 East Carson Street, Long Beach, CA 90822. Yale 1971. Subspecialty, gastroenterology. Board certified. Group, partnership, solo. Available August 1979.

Fred H.Miller, M.D., 378 Valley Park South, Bethlehem, PA 18018. Jefferson 1975. Board eligible. Group, partnership, solo. Available October 1979.

Rachakonda D. Prabhu, M.D., 80-15 41st Avenue, Apt. 4C8, Elmhurst, NY 11373. R. G. Kar Medical College (India) 1971. Subspecialty, pulmonary diseases. Board certified. Group, partnership, solo, or institutional. Available July 1979.

Samala R. Swamy, M.D., 355 Bard Avenue, Apt. 32, Staten Island, NY 10310. Kakatiya Medical College (India) 1972. Subspecialty, cardiovascular diseases. Board certified. Group, solo, or institutional. Available July 1979.

Jay H. Alexander, M.D., 30 Waterside Plaza, Apt. 9-E, New York, NY 10010. Loyola 1976. Subspecialty, emergency medicine. Board eligible. Industrial, emergency room, school health, institutional. Available July 1979.

Joel S. Steinberg, M.D., 6641 Oakland Street, Philadelphia, PA 19149. Temple 1976. Subspecialty, family practice. Board eligible. Group or partnership. Available July 1979.

N. Satish Kamath, M.D., 401 Tobin Drive, #310, Inkster, MI 48141. Karnatak Medical College (India) 1972. Board eligible. Group, partnership, solo. Available August 1979.

Sambaiah Kankanala, M.D., 122 Ashland Place, Apt. 11-N, Brooklyn, New York 11201. Osmania Medical School (India) 1972. Subspecialty, pulmonary diseases. Board certified. Group or solo. Available July 1979.

Nadakavil M. Koshy, M.D., 8 Slate Creek Drive, Apt. 9, Cheektowaga, NY 14227. Kottayam Medical College (India) 1971. Subspecialty, cardiovascular diseases. Board certified. Group, partnership, solo, or institutional. Available July 1979.

Peter K. Chak, M.D., 200 Bethel Loop, Apt. 20-D, Block B-2, Starret City, Brooklyn, NY 11239. Hong Kong University 1970. Board eligible. Any type practice. Available June 1979.

Chakumkal Cyriac,M.D., 3001 South Hanover Street, Baltimore, MD 21230. T.D. Medical College (India) 1973. Subspecialty, general practice. Board eligible. Group, partnership, solo. Available August 1979.

George T. Yang, M.D., 1764 Bising Avenue, Apt. 2, Cincinnati, OH 45239. Taipei (Taiwan) 1972. Subspecialty, endocrinology. Board certified. Group, partnership, solo, or emergency room. Available July 1979.

Pervaiz Rahman, M.D., 1650 Grand Concourse, Bronx, NY 10457. Dow Medical College (Pakistan) 1972. Subspecialty, gastroenterology. Board eligible. Group, partnership, institutional. Available July 1979.

Jambur E. Chandrashekar, M.D., 155 Forest Road, #39, St. John's A1A 1E7, Newfoundland, Canada. Mysore Medical College (India) 1970. Board eligible. Group, partnership, solo, or institutional. Available July 1979.

Jerome W. Takiff, M.D., 2917 E. Turquoise, Phoenix, Arizona 85028. Cornell 1973. Board certified. Group. Available.

Kenneth Lipkowitz, M.D., 76 E. Park St., Apt. 26, East Orange 07017. Guadalajara 1974. Board eligible. Partnership or group. Available July 1979.

N. M. Koshy, M.D., 8 Slate Creek Dr., Apt. 9, Cheektowaga, New York 14227. Cottyam-Kerala (India) 1971. Board certified. Subspecialty, cardiology. Group, partnership, solo, institution. Available July 1979.

Kiril Desai, 108-18 63rd Street, Forest Hills, New York 11375. Baroda (India). Subspecialty, cardiology. Group, partnership, salaried position. Available.

Vennaralagappan Mahadev, M.D., 429 Case Street, Kingsford, Michigan 49801. Madurai (India) 1967. Board certified. Subspecialty, cardiology. Group or solo. Available August 1979.

Harvey M. Cohen, M.D., 8 Deerwood West, Apt. 44C, Irvine, CA 92714. Syracuse 1972. Board certified. Subspecialty, gastroenterology. Solo, group, partnership. Available.

Taha Hamoui, M.D., 9831 Timerwood Circle, Louisville, KY 40223. Damascus (Syria). Board certified. Subspecialty, gastroenterology. Group, partnership, solo. Available July 1979.

Alan J. Sten, M.D., 105 West 13th St., Apt. 15-C, New York, NY 10011. New York Medical (1972). Subspecialty, infectious diseases. Board certified. Partnership or group. Available July 1979.

Pervaiz Rahman, M.D., 240 Yonkers Avenue, Apt. 1-C, Yonkers, NY 10701. Dow (Pakistan) 1972. Subspecialty, gastroenterology. Board eligible. Group, partnership, solo, or institutional. Available July 1979.

Aziz H. Junagadhwalla, M.D., 9 Nixon Court, Apt. 3-J, Brooklyn, NY 11223. Grant Medical College. Subspecialty, pulmonary medicine. Group or partnership. Available July 1979.

NEOPLASTIC DISEASES—Victor D. Ribeiro, M.D., 82 Beacon Hill, Apt. 1B3, Dobbs Ferry, NY 10522. Goa Medical College (India) 1970. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

Gregory P. Burke, M.D., 45 Viscount Road,Longmeadow, MA 01106. SUNY—Downstate 1972. Subspecialty, internal medicine. Board Certified (IM). Group or academic. Available.

NUCLEAR MEDICINE—R. Murty Krishnamsetty, M.D., 101 West 15th Street, Apt. 4-E North, New York, NY 10011. Banaras H. (India) 1971. Special interest, therapeutic radiology. Board eligible. Board certified in T.R. Group or partnership. Available July 1979.

OBSTETRICS/GYNECOLOGY—Khatoon T. Ginwala, M.D., 1817 Rolling Lane, Cherry Hill 08003. Hahnemann. Board eligible. Available July 1979.

Jeffrey M. Reinkraut M.D., 40-01 Little Neck Pkwy., Little Neck, NY 11363. Rutgers (CMDNJ) 1975. Board eligible. Group or partnership. Available July 1979.

Kok C. Chang, M.D., 411 West 114th Street, Apt. 4-B, New York, NY 10025. National Taiwan University 1973. Board eligible. Group, partnership, solo. Available August 1979.

Nilima B. Parekhji, M.D., 12000 Fairhill Road, Apt. 814, Cleveland, OH 44120. T.N. Medical College (India) 1971. Board eligible. Group, partnership. Available July 1979.

Devendra M. Bhardwaj, M.D., 242 Gurdon Street, Bridgeport, CT 06606. S.M.S. Medical College (India). Board eligible. Group, partnership, solo, HMO. Available July 1979.

Kenneth Garrett, M.D., 31-A1 Lakeshore Drive, Farmington, CT 06032. NYU 1975. Board eligible. Group, partnership. Available August 1979.

Charles Ackerman, M.D., 353 East 17th Street, New York, NY 10003. Tel Aviv (Israel) 1975. Board eligible. Group or partnership. Available September 1979.

Sang Bak Yoo, M.D., 10856 Nottingham, Detroit, MI 48224. Kyung-Pook University (Korea) 1969. Board eligible. Group, partnership, or solo. Available July 1979.

Steven B Wirts, M.D., 340 Nina Street, New Windsor, NY 12550. Jefferson 1974. Board eligible. Group or partnership. Available July 1979.

OCCUPATIONAL MEDICINE—Arthur E. Mittnacht, M.D., 299 Edgerstoune Road, Princeton 08540. Columbia 1945. Subspecialty, orthopedic surgery. Board certified (orthopedic surg.) Industrial, school health, emergency room. Available May 1979.

OPHTHALMOLOGY—Jonathan F. Wise, M.D., 1175 York Avenue, Apt. 4-H, New York, NY 10021. SUNY—Buffalo 1973. Subspecialty, pediatrics. Board eligible. Group or partnership. Available June 1979.

Joshua Frankel, M.D., 161 North Sycamore Avenue, Los Angeles, CA 90036. Maryland 1971. Board certified. Partnership, solo, institutional. Available June 1979.

Alan I. Mandelberg, M.D., 284 Wintergreen Way, Rochester, NY 14618. SUNY—Buffalo 1972. Board certified. Group, partnership, or solo. Available July 1979.

Raphael J. Sapeika, M.D., 7 Windsor Road, Great Neck, NY 11021. Cape Town (South Africa) 1970. Board eligible. Group or partnership. Available July 1979.

Stephen L. Bosniak, M.D., 782 Westend Avenue, Apt. 63, New York, NY 10025. Georgetown 1973. Board elilible. Group, partnership, solo, institutional, or industrial. Available June 1979.

OTORHINOLARYNGOLOGY—Jang-Huei Jang, M.D., 7563 Drumheller Drive, Richmond, Virginia 23225. Chung-Shan (Taiwan) 1967. Board eligible. Group or solo. Available July 1979.

Gregory J. Milmoe, M.D., 259 Canner Street, New Haven, CT 06511. Chicago 1973. Board eligible. Group, research, partnership. Available August 1979.

PATHOLOGY—Mansoora R. Chaudry, M.D., 41-04 Goldblatt Terrace, Fair Lawn 07410. Fatima Jinnah Medical College (Lahore, India). Board certified. Any type practice. Available.

Dhanasaran Mongkolsmai, M.D., 900 West Spencer Street, Apt. 302, Philadelphia, PA 19141. Ramathibodi (Thailand)

1971. Special interest, clinical pathology. Board certified. Group, partnership, institutional. Available September 1979.

Ahm Abdul Mannan, M.D., 215 East 77th Street, Apt. 3-C, New York, NY 10021 Dacca (Pakistan) 1961. Special interest, clinical pathology. Board eligible. Group, research, institutional. Available July 1979.

Luis R. Molinas, M.D., 3041 West Chester Pike, Apt. G-7, Broomall, PA 19008. National University (Paraguay) 1972. Board eligible. Partnership, group, institutional. Available July 1979.

Ruly Yoediono, M.D., 10304 East Malcolm Circle, Cockeysville, MD 21030. Padjadjaran (Bandung) 1971. Special interest, clinical pathology. Board eligible. Group, partnership. Available September 1979.

Mehboob M. Fatteh, M.D., 3405 Crane Ferry Road, Augusta, GA 30907. H.H.L. Medical College (India) 1968. Board eligible. Group, partnership, solo, institutional. Available March 1979.

Frank R. Rudy, M.D., 7614 Woodcrest Avenue, Philadelphia, PA 19151. Pittsburgh 1974. Board eligible. Institutional, partnership, research. Available July 1979.

PEDIATRICS—Georgino R. Reyes, M.D., 18 Palmetto Circle, Brownsville, TX 78521. Santo Domingo (Dominician Republic) 1960. Board certified. Group, partnership, research. Available July 1979.

Jesse M. Mintz, M.D., 265-24 74th Avenue, Apt. G-1, Glen Oaks, NY 11004. Columbia 1972. Board certified. Institutional, group. Available July 1979.

Ganesh N. Deshpande, M.D., 49 Stoneledge Court, Williamsville, NY 14221. Grant Medical College (India) 1970. Subspecialty hematology. Board eligible. Institutional, research, group. Available July 1979.

Chalermlarp Mongkolsmai, M.D., 900 West Spencer Street, Apt. 302, Philadelphia, PA 19141. Ramathibodi Medical School (Thailand) 1971. Subspecialty, pediatric cardiology. Board certified. Institutional, research, group. Available September 1979.

Theodore H. Sher, M.D., 170 West End Avenue, New York, NY 10023. SUNY—Downstate 1974. Subspecialty, pediatric allergy. Board eligible. Group, partnership. Available July 1979.

Nam Soo Lee, M.D., Meadowbrook Apt. 44, Blessing Road, Slingerlands, NY 12159. Seoul National University (Korea) 1971. Subspecialty, pediatric cardiology. Board eligible. Solo, group, research. Available July 1979.

Warren P. Silberstein, M.D., 17 Bartlett Drive, Middletown, NY 10940. SUNY—Downstate 1974. Board eligible. Group, institutional, emergency room, school health. Available July 1979.

Elias G. Gouel, M.D., 6036 East Pratt, Baltimore, MD 21224. Ain Shams University (Egypt) 1971. Board eligible. Group, partnership. Available August 1979.

Mehboob A. Sachani, M.D., 1 Slate Creek Road, Apt. 3, Cheektowaga, NY 14227. Seth G.S. Medical College (India) 1974. Board eligible. Group or solo. Available July 1979.

Ira B. Toback, M.D., 4618 Avenue J. Brooklyn, NY 11234. Louvain (Belgium) 1976. Board eligible. Partnership or group. Available July 1979.

Bart Asner, M.D., 3949 Los Feliz Blvd., Los Angeles, CA 90027. Pennsylvania 1976. Group or partnership. Available July 1979.

Cecil F. Desai, M.D., 2112 Starling Ave., #5K, Bronx, NY 10462. B.J. Medical College (India) 1968. Board eligible. Salaried position. Available July 1979.

M.S. Krishna, M.D., 32127 Concord Dr., Bldg. 6, Apt. F, Madison Heights, Michigan 48071. Bangalore (India) 1971. Board eligible. Group or partnership. Available July 1979.

PHYSICAL MEDICINE/REHABILITATION—Diane C. Griffin, M.D., 29 East 29th Street, New York, NY 10016. University of Rome (Italy) 1974. Subspecialty, general practice Board eligible. Institutional, group, research. Available July 1979.

PSYCHIATRY—Sumanlal J. Kaneria, M.D., P.O. Box 9587, Cheyenne, WY 82001. M.P. Shah Medical School (India) 1971. Board eligible. Academic type practice. Available October 1979.

Balasubramaniam Gulasekaram, M.D., Apt. 1-D, Beechwood Hall, Westchester City Medical Center, Valhalla, NY 10595. Colombo (Sri Lanka) 1970. Board eligible. Research, institutional, group. Available September 1979.

Herman Berliss, M.D., 110 Boteler Street, Apt. 703, Ottawa, Ontario K1N 8W8. Canada. Case Western Reserve 1974. Board eligible. Adult outpatient. Available July 1979.

PULMONARY DISEASES—Shreedhar B. Shetty, M.D., 1168 Davey Street, Bloomfield 07003. Kasturbha Medical College (India) 1967. Subspecialty, internal medicine. Board eligible. Group, institutional, industrial. Available July 1979.

Joseph I. Matthews, M.D., OSC Box 553, Walter Reed Hospital, Washington, DC 20012. Creighton 1969. Subspecialty, internal medicine. Board certified (IM). Group, institutional, partnership. Available August 1979.

Richard T. Bell, M.D., 1740 Aidenn-Lair Road, Dresher, PA 19025. Jefferson 1972. Subspecialty, internal medicine. Board certified (IM). Group, solo. Available July 1979.

Harjinder S. Gogia, M.D., 6201 North 10th Street, Apt. 435, Philadelphia, PA 19141. Punjabi University (India) 1973. Subspecialty, internal medicine. Board certified (IM). Group, institutional. Available July 1979.

RADIOLOGY—Seetharam A. Mattupalli, M.D., 7083 Middle Valley Walk, Apt. B, St. Louis, MO 63123. Guntur (India) 1971. Subspecialty, diagnostic radiology. Board eligible. Partnership, research, group, administrative, institutional. Available July 1979

Pongthep Thepsongwajja, M.D., 2309 NW 38th Street, Apt. 75, Lawton, Oklahoma 73505. Siriraj (Thailand) 1972. Board certified. Group, partnership, solo. Available.

R. Murty Krishnamsetty, M.D., 101 West 15th Street, Apt. 4-E North, New York, NY 10011. Banaras H. (India) 1971. Subspecialty, nuclear medicine. Board certified (therapeutic radiology). Board eligible (N.M.) Group, partnership. Available July 1979.

RHEUMATOLOGY—Carlos Ganuza, M.D., 2D Beaver Court, Wilkes-Barre, PA 18702. University of El Salvador 1971. Subspecialty, internal medicine. Board eligible (IM). Institutional, group. Available July 1979.

Stuart P. Brogadir, M.D., 6357 Lancaster Avenue, Philadelphia, PA 19151. Cornell 1975. Subspecialty, internal medicine. Board eligible (IM). Research, group. Available July 1979.

SURGERY, CARDIOVASCULAR—Michael L. Weinberger, M.D., 5902 Weatherford Road, Fayetteville, NC 28303. SUNY—Downstate 1971. Subspecialty, thoracic surgery. Board eligible (general surgery). Group or partnership. Available June 1979.

SURGERY, GENERAL—Clementino O. Purisima, M.D., 105 Chadwick Road, Teaneck 07666. Santo Tomas (Philippines) 1969. Board eligible. Group partnership, solo. Available July 1979.

John L. Deboer, M.D., 1242B Cedar Street, Fort Dix 08640. Vermont 1971. Board certified. Group or partnership. Available June 1979.

Joel A. Yellin, M.D., 215-16 15th Road, Bayside, New York 11360. Rochester 1973. Board eligible. Partnership, solo, group. Available July 1979.

Glenn A. Pasternack, M.D., 7660 Phoenix Drive, Apt. 1559, Houston, TX 77030. SUNY—Downstate 1974. Board eligible. Partnership, group. Available July 1979.

Marc A. Grossman, M.D., 21140 18th Avenue, Bayside, NY 11360. Einstein 1975. Board eligible. Partnership or group. Available July 1979.

Dilipkumar H. Kakaiya, M.D., Lafayette Towers, Apt. 709, 2040 Lehigh Street, Easton, PA 18042. Grant Medical College (India) 1971. Board eligible. Solo, partnership, group. Available July 1979.

Bassam T. Kashlan, M.D., 65 Cedar Avenue, Apt. D-16, Long Branch 07740. Damascus (Syria) 1972. Board eligible. Partnership, institutional, group. Available July 1979.

Frederick J. Heinle, Jr., M.D., 328 S. Linden Avenue, Pittsburgh, PA 15208. Board eligible. Partnership, group, solo. Available July 1979.

Stephen F. Winer, M.D., Box 633, Wiesbaden, West Germany, APO New York 09220. Wayne 1967. Board certified. Partnership, group. Available January 1980.

Meherwan J. Mistry, M.D., 524 Morris Avenue, Apt. 3-E, Elizabeth 07208. Grant Medical College (India) 1972. Board eligible. Partnership, group, solo. Available July 1979.

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SURGERY, GENERAL—Ivan A. Shulman, M.D., 785 Burnett Avenue, Apt. 7, San Francisco, CA 94131. Pittsburgh 1972. Board eligible. Group, partnership. Available July 1979.

Donald F. Temple, M.D., 44 Morningside Dr., Apt. 5, New York 10025. Cornell 1974. Board eligible. Group, partnership, hospital, solo. Available July 1979.

SURGERY, ORTHOPEDIC—Stefan Glowacki, M.D., 2130 Williamsbridge Road, Bronx, NY 10461. Warsaw (Poland) 1962. Subspecialty, orthopedic surgery. Board eligible (orth. surg.) Solo, partnership, research. Available July 1979.

Ronald J. Leavitt, M.D., 717 Willow Way, Narberth, PA 19072. Columbia 1971. Subspecialty, hand surgery. Board certified. Partnership, group. Available July 1979.

Samir R. Dawoud, M.D., 1120B Easton Avenue, Bldg. 18, Somerset 08873. Kasr El Aini (Egypt) 1960. Board eligible. Partnership, group. Available July 1979.

Lawrence Schweitzer, M.D., 23 Woodward Crescent, Halifax, Nova Scotia B3M 1J6. Louvain 1975/Dalhousie 1979. Board eligible. Group, partnership, solo. Available July 1979.

T. Ghavamian, M.D., P.O. Box 3452, San Diego, CA 92103. Teheran 1953. Board certified. Group, partnership, solo.

SURGERY, UROLOGICAL—Robert J. Claire, M.D, 111 Annadale Road, Staten Island, NY 10312. Guadalajara (Mexico) 1974. Special interest in family counseling. Any type practice. Available July 1980.

Natvarlal P. Patel, M.D., 404 Sixth Stteet, Apt. 5D, Coraville, IA 52241. B. J. Medical School (India) 1970. Subspecialty, general surgery. Board eligible. Solo, partnership, group, research. Available July 1979.

Daniel H. Piazza, M.D., 741 Peninsula Court, Ann Arbor, MI 48105. Illinois 1972. Board eligible. Group or partnership. Available July 1979.

Sheldon Muhlbauer, M.D., 3450-23 Wayne Avenue, Bronx, NY 10467. Einstein 1973. Board eligible. Partnership, group. Available July 1979.

Shree Subhash, M.D., 8 Sugarloaf Court, Baltimore, MD 21209. Prince of Wales Medical College (India) 1967. Partnership, group, solo. Available September 1979.

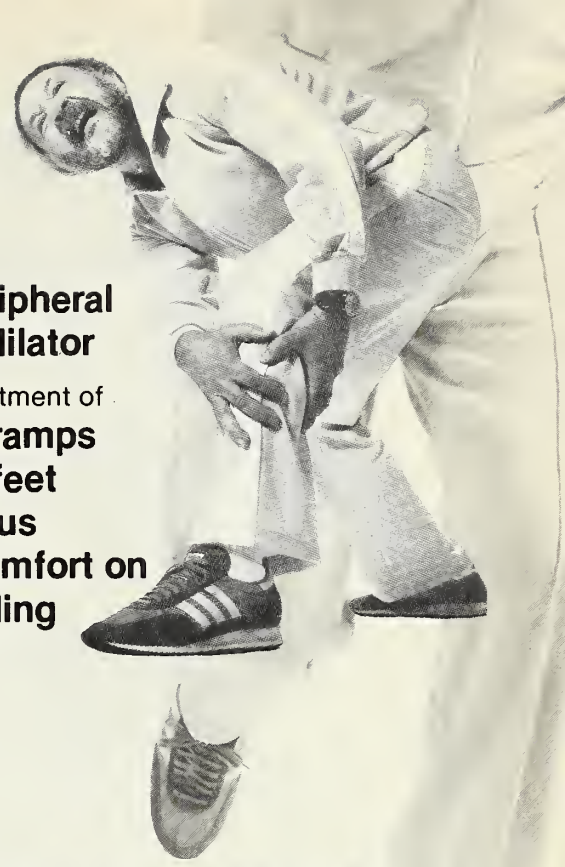
Report from the Foundation

Daniel J. O'Regan, M.D.
Medical Director

Mr. Howard R. Veit, Director of the Health Maintenance Organization Program of HEW, addressed a national HMO policy conference in Washington in February. He projected the targeted development strategy for the next few years. Program dollars (federal dollars) best will be used by focusing on "maximizing the cost savings potential of HMOs." The "targets" for development include: (1) Locations with the largest populations, highest health care costs, and the fastest growing populations; (2) Engendering maximum competition among HMOs. This includes group models, staff models, and IPA models. It also will compare prepaid care with traditional medical delivery modes; (3) The encouragement of established HMOs to move into new or expanded service areas; and (4) Maximum possible enrollment in the targeted areas. The focus will be on 61 target areas over the next five years. These are SMSAS (Standard Metropolitan Statistical Areas) with over 250,000 population whose market penetration rate for HMOs is under six percent. The highest health cost areas have top priority. Next will be the highest growth areas, and third will be areas with "above average" health care costs.

The commitment is there for expansion of prepaid care. The emphasis of many planners leans toward the closed panel models. "Fee-for-service" is a phrase not found in the vocabulary of such individuals. Prepaid health *versus* "fee-for-service" is often the attitude. Your Foundation continues to emphasize the fee-for-service IPA mode of prepaid health. For a good discussion of these terms, review the recent JAMA article by Dr. Egdahl and associates (Egdahl, R.H., *et al:* Fee-for-service health maintenance organizations. *JAMA* 241:588-591, 1979).

Projections for the future are sometimes nebulous exercises. As we prepare for the decade of the eighties, it is clear that *some* form of prepaid medical care will exist. The extent of penetration of the market remains to be seen. There will be thrusts by "liberals" to use prepaid systems as a part of national health insurance. There also will be activity by "conservatives" to preserve fee-for-service traditions. The Individual Practice Association mode, with its fee-for-service component, should be allowed to find a substantial part of the "market."



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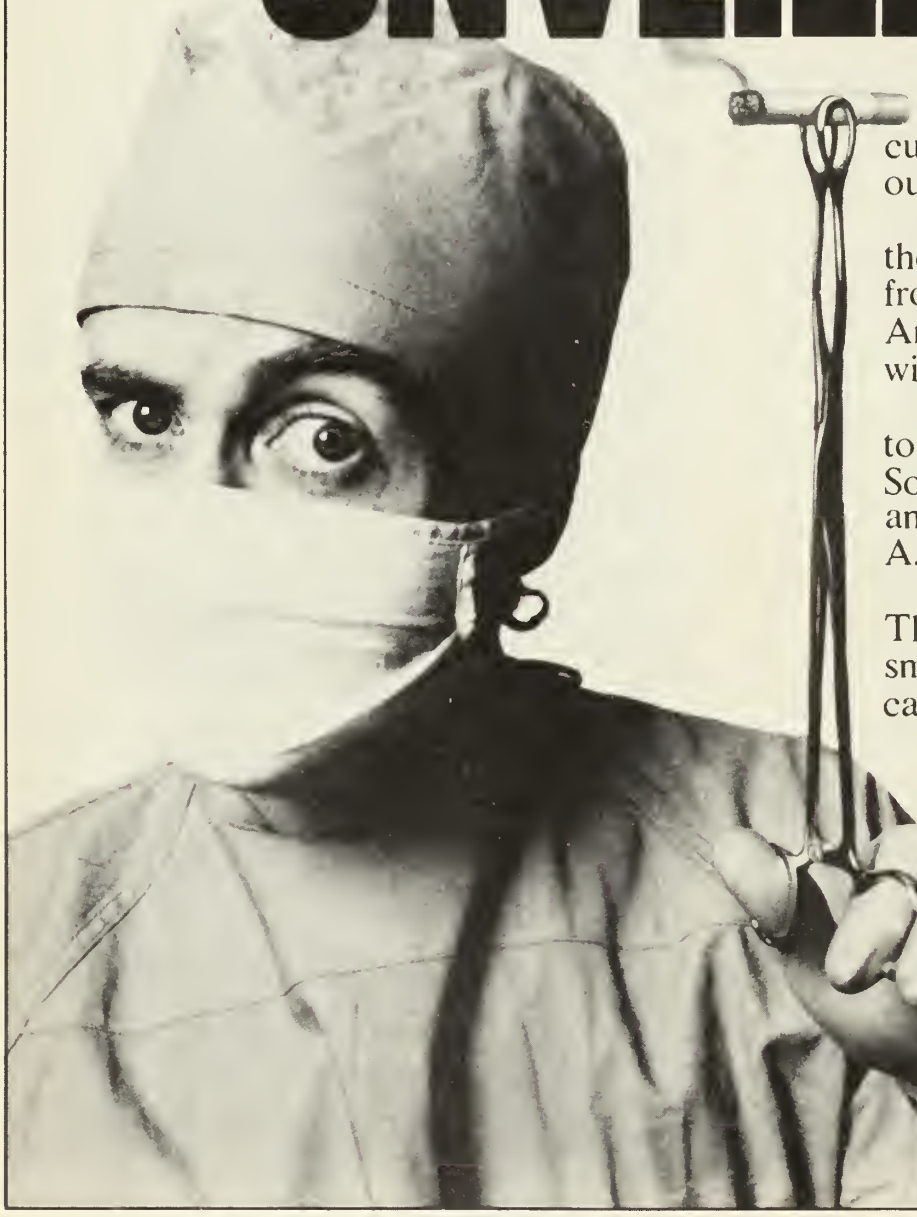
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NUTRITION UPDATE—1979

Third Annual Nutrition Symposium
April 26, 27, 1979

Dunlap Auditarium, Medical Educatian Building
36th & Hamiltan Walk, Philadelphia, Pa. 19104

Course Director:

James L. Mullen, M.D.
Chief Surgery, Philadelphia VA Medical Center
Director, Nutrition Support Service, Hospital of the University of Pennsylvania
Assistant Professor of Surgery, University of Pennsylvania School of Medicine

The purpose of this course is to provide the practicing physician—pharmacist—nurse—clinical nutritionist—hospital administrator—with a physiologically-based clinical approach to commonly encountered diagnostic and therapeutic problems in clinical nutrition. The emphasis throughout will be on the clinically important and practical aspects of the topics to be discussed. The course will be suitable for a wide range of clinicians—physicians, clinical nutritionists, nurses and pharmacists. The workshops will emphasize the day-to-day practical aspects of nutritional support with audience participation and educational benefit.

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WHERE: The Medical College of Pennsylvania
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University of Maryland
School of Medicine
10 S. Pine Street
Baltimore, Maryland 21201
(301) 528-3956

CME CALENDAR

This listing is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, the Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the College of Medicine and Dentistry of New Jersey. For information on accreditation, please contact the sponsoring organization(s), indicated by italics—last line of each item.

MEDICINE (includes Family, Internal, and General Medicine and Dermatology)

May

1 Outpatient Procedures
29 Spondyloarthropathy
8-9 a.m.—Garden State Community Hospital, Route 73, Marlton
(Garden State Community Hospital and AMNJ)

1 Asthma, Allergy and Pulmonary Medicine
8:30 a.m.-1 p.m.—Holy Name Hospital, Teaneck
(Holy Name Hospital, National Jewish Hospital for Chest Diseases and Bergen-Passaic Lung Association)

2 Dinner Meeting
6:30-9:30 p.m.—Holiday Inn, East Orange
(AMNJ Endocrinology Section)

2 New Developments in Scanning
30 Cerebral Vascular Disease
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

2 Medical Grand Rounds
11:30 a.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

2 Endocrine Conferences
9 3:30-5 p.m.—Rotates between Newark
16 Beth Israel Medical Center, Martland
23 Hospital, Newark, and VA Hospital,
30 East Orange
(AMNJ Endocrinology Section)

2 Grand Rounds and Case Presentations
9 2-4 p.m.—Rotating between Martland
16 Hospital, Newark Beth Israel, St.
23 Michael's, St. Joseph's Hospitals and
30 Jersey City Medical Center
(CMDNJ and AMNJ)

2 Advances in Medicine
9 9:30 a.m.-11 a.m.—Bergen Pines
16 County Hospital, Paramus
23 *(Bergen Pines County Hospital and*
30 *AMNJ)*

2 Allergy: Asthma
9 Acute Renal Failure
16 Medical Lecture Series
23 1-3 p.m.—Christ Hospital, Jersey City
30 *(Christ Hospital and AMNJ)*

3 Cardiology Conferences
10 2:15-4:15 p.m.—Deborah Heart and Lung
17 Center, Browns Mills
24 *(Deborah Heart and Lung Center and*
31 *AMNJ)*

4 Interpretation of Renal Function Tests
9 Renal Diseases and Pregnancy
11 IVP in Ultrasound and Renal Disease
18 Acid Base Imbalance
26 Chronic Renal Failure
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

4 Coronary Artery Disease
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

8 Virology
4-6 p.m.—Institute for Medical Research, Copewood St., Camden
(Institute for Medical Research and AMNJ)

8 Mycosis Fungoides
8-10 p.m.—Schering Corporation, Kenilworth
(NJ Dermatological Society and AMNJ)

8 Gram Negative Sepsis
8:30-9:30 p.m.—Riverside General Hospital, Secaucus
(Riverside General Hospital and AMNJ)

9 Surgically Corrected Congenital Heart Disease
12:45-2 p.m.—St. Francis Medical Center, Trenton
(American Heart Association and AMNJ)

9 Dietary Enhancement of CNS Neurotransmitters
16 Hazards of Polypharmacy in Psychiatry
23 Calcium-Phosphorus Homeostasis and Metabolic Bone Disease
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

9 Annual Awards Dinner, Academy of Medicine of New Jereey
6 p.m.—The Chanticler, Millburn
(AMNJ)

10 Case Presentations and Guest Speakers
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

10 Regional Chest Conferences
7:30-9:30 p.m.—The Valley Hospital
(NJ Thoracic Society and AMNJ)

13 Annual Meeting
8 a.m.-12 noon—Atlantic City
(Oncology Society of NJ and AMNJ)

15 Lung Cancer
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

17 Current Therapy for Sexual Dysfunction
5-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

19- Recent Advances in Laboratory Medicine
20 9 a.m.-4:30 p.m.—Metpath Institute for Medical Education, 60 Commerce Way, Hackensack
(Metpath Institute for Medical Education and AMNJ)

22 Suicidology
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

23 Interstitial Pulmonary Disease
11:30 a.m.-1 p.m.—VA Hospital, East Orange
(VA Hospital and AMNJ)

23 Physical Medicine and Rehabilitation for the Family Practitioner
8:30 a.m.-1 p.m.—John F. Kennedy Medical Center, Edison
(AMNJ)

23 Low Back Pain
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

29 Drug Therapy of COLD
8-9 a.m.—Greater Paterson General Hospital, Wayne
(Greater Paterson General Hospital and AMNJ)

30 Cardiac Grand Rounds
3-4:30 p.m.—NJ Medical School, Newark
(American Heart Association, NJ Affiliate, and AMNJ)

June

1 Adrenal Diseases
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

5 Weekly Tumor Conferences
12 11 a.m.-12 noon—Morristown
19 Memorial Hospital
26 *(Morristown Memorial Hospital and*
AMNJ)

6 Advances in Medicine
13 9:30 a.m.-11 a.m.—Bergen Pines County
20 Hospital, Paramus
(Bergen Pines County Hospital and AMNJ)

6 Hypoglycemia
13 Diseases of the Esophagus
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

6 Endocrine Conferences
3:30-5 p.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, E.O.
(AMNJ Endocrinology Section)

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July 13-15 Anaheim, CA	July 13-15 Orlando, FL	July 13-15 Great George, NJ	
August 17-19 Toronto, Canada	July 27-29 Lake Geneva, WI	July 27-29 Monterey, CA	
August 24-26 Hilton Head, SC	August 17-19 Cape Cod, MA	August 10-12 Shanty Creek Resort, MI	
September 28-30 Las Vegas, NV	September 28-30 San Francisco, CA	August 17-19 Orlando, FL	

- For information on these and other courses, contact: Director of CME, Dept. 7, International Medical Education Corporation 64 Inverness Drive East, Englewood, Colorado 80112/Tel: (303) 773-1144 or Toll Free (800)525-8646, ext 236

6 **Clinical Shock**
11:30 a.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

6 **Echocardiography**
13 **Psychotropic Medication**
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

6 **Diabetic Coma and Pre and Post-operative Diabetic Care**
13 **Medical Lecture Series**
20 1-3 p.m.—Christ Hospital, Jersey City
27 *(Christ Hospital and AMNJ)*

7- **Lectures**
10 Southampton Princess Hotel, Bermuda
(NJ Dermatological Society and AMNJ)

7 **Anemia**
12 **Anemia in Pregnancy**
14 **Leukemia**
21 **Blood Disease Tests**
28 **Drug Interactions**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

7 **Cardiology Conferences**
14 2:15-4:15 p.m.—Deborah Heart and
21 Lung Center, Browns Mills
28 *(Deborah Heart and Lung Center and AMNJ)*

12 **Lower Back Pain**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

14 **Case Presentations and Guest Speakers**
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

19 **Peripheral Vascular Disease**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

19 **Cardiac Arrhythmias**
8:30 p.m.—Omar's Restaurant, Fair Lawn
(Fair Lawn Memorial Hospital and AMNJ)

21 **Regional Chest Conferences**
7:30-9:30 p.m.—Newark Beth Israel Medical Center
(NJ Thoracic Society and AMNJ)

21 **Infectious Diseases**
4-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

26 **Scanning, New Developments**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

27 **Cardiac Grand Rounds**
3-4:30 p.m.—CMDNJ-NJ Medical School
(American Heart Association, NJ Affiliate and AMNJ)

NEUROLOGY/PSYCHIATRY

May

1 **Seminar on Law and Psychiatry**
8 3:30-5:30 p.m.—Rutgers Law School,
15 Newark
22 *(Rutgers University Law School and*
29 *AMNJ)*

2 **Personality Disorders**
11:30-12:30 p.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

2 **Course for Psychiatrists**
9 8-10 p.m.—Hackensack Hospital
(NJ Psychoanalytic Society and AMNJ)

2 **Psychology of Women—Research**
16 **Psychiatric Diagnosis Values**
30 **Physicians as Patients and Their Families**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

3 **Improving Psychotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, South Orange
(Group for Advanced Psychiatric Study and AMNJ)

3 **Psychiatric Lecture Series**
10 11 a.m.-12 noon—Greystone Park
17 Psychiatric Hospital
24 *(Greystone Park Psychiatric Hospital and AMNJ)*

6 **Treatment of Sexual Dysfunction**
9 a.m.-5 p.m.—Ramada Inn, Rochelle Park
(The Center for Sexual and Relationship Development and AMNJ)

7 **Depression in an Adolescent Girl**
8-10 p.m.—1046 South Orange Avenue, Short Hills
(Essex Psychiatric Seminar and AMNJ)

7 **Neuroscience Conferences**
14 11:30 a.m.-12:30 p.m.—Bergen Pines
21 County Hospital, Paramus
(Bergen Pines County Hospital and AMNJ)

9 **Psychotropic Medication**
1:30-3:30 p.m.—Trenton Psychiatric Hospital
(Trenton Psychiatric Hospital and AMNJ)

9 **Suicidology**
1:30-2:30—John E. Runnells Hospital of Berkeley Heights
(John E. Runnells Hospital and AMNJ)

16 **Child Rearing Implications from Group Dynamic Studies**
30 **Non-Verbal Communication**
1:30-3 p.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

June

4 **Anorexia Nervosa in an Adult Married Woman**
8-10 p.m.—9 Marquette Road, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

6 **Depression-Outpatient Treatment**
13 **Psychodynamic Therapy for Outpatients**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

7 **Improving Psychotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, So. Orange
(Group for Advanced Psychiatric Study and AMNJ)

13 **Unusual Psychiatric Disorders and Atypical Psychoses**
1:15-2:45 p.m.—Marlboro Psychiatric Hospital
(Marlboro Psychiatric Hospital and AMNJ)

13 **Psychiatry in the U.S.**
1:30-3 p.m—NJ Medical School, Newark
(CMDNJ and AMNJ)

14 **Lecture Series**
8:30-10:30 p.m.—Hackensack Hospital
(NJ Psychoanalytic Society and AMNJ)

19 **Clinical Shock**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

OBSTETRICS/GYNECOLOGY

May

2 **Lectures in Obstetrics and Gynecology**
8-10 a.m.—Location varies
(CMDNJ and AMNJ)

2 **Pros and Cons of Estrogen Replacement Therapy**
9-11 a.m.—Middlesex General Hospital, New Brunswick
(Middlesex General Hospital, AMNJ and AAFP)

3 **Grand Rounds and Case Presentations**
10 4-5 p.m.—Martland Hospital, Newark
17 *(CMDNJ and AMNJ)*
24
31

9 **Breast Diseases in Gynecologic Practice**
8 a.m.-2 p.m.—Garden State Community Hospital, Marlton
(Garden State Community Hospital and AMNJ)

15 **Breast Cancer**
7-8 p.m.—Irvington General Hospital
(Irvington General Hospital and AMNJ)

15 **Breast Cancer**
22 **Current Chemotherapy**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

June

6 **Grand Rounds and Case Presentations**
13 2-4 p.m.—Rotating between Martland
20 Hospital, Newark Beth Israel, St.
27 Michael's, and St. Joseph's Hospitals, Newark, and Jersey City Medical Center
(CMDNJ and AMNJ)

7 **Grand Rounds and Case Presentations**
14 4-5 p.m.—Martland Hospital, Newark
21 *(CMDNJ and AMNJ)*
28

Gynecologic Endoscopy
14 8 a.m.-5 p.m.
15 7:45 a.m.-4:30 p.m.
Memorial General Hospital, Union
(New Jersey Fertility Foundation and AMNJ)

PATHOLOGY/CLINICAL PATHOLOGY

May

2 **Clinical Pathology Grand Rounds**
12 noon-1 p.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

23 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

June

6 **Clinical Pathology Grand Rounds**
13 12 noon-1 p.m.—NJ Medical School,
20 Newark
(CMDNJ and AMNJ)

27 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

PEDIATRICS

May

4 **Advances in Pediatrics**
11 9:30-10:30 a.m.—NJ Medical School,
18 Newark
25 *(CMDNJ and AMNJ)*

24 **Pediatric and Adolescent Gynecology**
8:30-10 p.m.—179 So. Maple Ave., Ridgewood
(American Medical Women's Association and AMNJ)

RADIOLOGY

May

7 **Use of Body Scan in Surgical Patients**
7:45-9 a.m.—Newark Beth Israel Medical Center
(Newark Beth Israel Medical Center and AMNJ)

8 **Upper Extremity Trauma**
8-9 a.m.—Garden State Community Hospital, Route 73, Marlton
(Garden State Community Hospital and AMNJ)

16 **Advances in X-Ray Diagnosis and Treatment**
1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

16 **Dinner Meeting**
6:30 p.m.—The Manor, West Orange
(Radiotherapy Section, AMNJ)

17 **Seminar**
7:15-10:15—Hospital Center at Orange
(Radiological Society of NJ and AMNJ)

GENERAL SURGERY

May

1 **Weekly Tumor Conferences**
8 11 a.m.-12 noon—Morristown
15 Memorial Hospital
22 *(Morristown Memorial Hospital*
29 *and AMNJ)*

7 **Lecture Series in Surgery**
14 4:30-5:30 p.m.—NJ Medical School,
21 Newark
(CMDNJ and AMNJ)

SURGICAL SPECIALITIES (includes ENT, neurosurgery, ophthalmology, orthopedics, plastics, and vascular surgery)

May

2 **Neurovascular Surgery**
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

3 **Neurosurgical Case Presentations**
10 4-5:30 p.m.—NJ Medical School,
17 Newark
24 *(CMDNJ and AMNJ)*
31

4 **Orthopedics**
11 7:30-9 a.m.—Alexian Brothers Hospital,
15 Elizabeth
(Alexian Brothers Hospital)

16 **Contact Lens Treatment**
7:15-9:30 p.m.—United Hospitals Medical Center, Annex Bldg., Newark
(Associated Eye Residencies of NJ and AMNJ)

22 **Plastic Surgery in Salvaging Lower Extremity**
8-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

June

1 **Orthopedics**
8 7:30-9 a.m.—Alexian Brothers Hospital,
22 Elizabeth
(Alexian Brothers Hospital

7 **Neurosurgical Case Presentations**
14 4-5:30 p.m.—NJ Medical School,
21 Newark
(CMDNJ and AMNJ)

20 **Management of Face Turns with Nystagmus**
7:15-9:30 p.m.—United Hospitals Medical Center, Annex Bldg., Newark
(Associated Eye Residencies of NJ and AMNJ)

26 **Cervical Spine Injuries**
6-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

MISCELLANEOUS

May

8 **Malpractice**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

9 **Suicidology**
1:30 p.m.—John E. Runnells Hospital, Berkeley Heights
(John E. Runnells Hospital and AMNJ)

9 **Annual Awards Dinner**
6 p.m.—Chanticler, Millburn
(AMNJ)

13 **Annual Meeting**
8 a.m.-12 noon—Atlantic City
(Oncology Society of NJ and AMNJ)

June

26 **PSRO**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

OBITUARIES

Dr. Robert Bozzi

We have just learned of the death on January 26 of Robert Bozzi, M.D., of Newark. Born in New Haven, Connecticut in 1904, Dr. Bozzi earned his medical degree from the Medical College of the University of Naples (Italy) in 1936. He was a general practitioner and had been on the staff at the Columbus Hospital in Newark and the Clara Maass Memorial Hospital in Belleville. During World War II, he served for four years with the medical department of the AUS.

Dr. Leonard P. Burke

On January 23, Leonard P. Burke, M.D., a senior member of our Essex County component, died at his home. Born in 1902 in Minneapolis, Dr. Burke was graduated from the University of Minnesota School of Medicine in 1931 and pursued a residency in family medicine. He practiced in Verona for many years until retirement in 1975. Dr. Burke had been affiliated with the Community and St. Vincent's Hospitals in Montclair and with the Jersey City Medical Center. He was a member of the Academy of Medicine of New Jersey.

Dr. Vincent J. Felitti

Vincent J. Felitti, M.D., a member of our Hudson County component, died suddenly on January 2 in San Diego, California. A graduate of the Medical College of the University of Virginia, class of 1927, Dr. Felitti had practiced pediatrics in Hudson County for fifty years before retiring to California in

1976. He had been attending pediatrician at St. Mary's Hospital in Hoboken and New York University Hospital, and was a member of the faculty at the New York Postgraduate Medical School. Dr. Felitti was 82 years old at the time of his death. A son, Vincent J. Felitti, Jr., M.D., is a practicing physician in San Diego.

Dr. Emerson S. Haines

The former head of the department of anesthesiology at the Jersey Shore Medical Center, Emerson S. Haines, M.D., died there on February 5. Born in Downingtown, Pennsylvania in 1899, Dr. Haines was graduated from Hahnemann Medical College in 1929, first having been graduated from the Philadelphia College of Pharmacy. He practiced general medicine in Asbury Park for 14 years and then took graduate training in anesthesiology, which he pursued full time at Jersey Shore Medical Center in Neptune until retiring in 1969. He was a Fellow of the American College of Anesthesiologists and a member of the American and New Jersey Societies of Anesthesiology. During World War II, Dr. Haines served three years with the medical department of the AUS.

Dr. George Heller

A prominent Bergen County pediatrician, George Heller, M.D., died on January 31. Born at the turn of the century, Dr. Heller earned his medical degree from Johns Hopkins University Medical School in 1925 and pursued a residency in pediatrics at Vanderbilt Hospital in Nashville. He came to Englewood to establish a pediatric practice in 1928, and remained there until retirement in the early 1970s. Dr. Heller was board certified in his chosen field and a Fellow of the American Academy of Pediatrics, and had been chief of pediatrics and chief of staff at the Englewood Hospital. He was president of the Bergen County Medical Society in 1958-1959 and was the author of several published articles in his specialty. During World War II, Dr. Heller served four years with the Department of Medicine of the AUS in the Pacific theater. He was a 1975 recipient of MSNJ's Golden Merit Award indicating fifty years of medical practice.

Dr. Lee R. Herrington

Word has just been received of the death in Englewood Hospital on December 17, 1978, of Lee R. Herrington, M.D., after a prolonged illness. Born in Texas in 1904, Dr. Herrington was graduated from Temple University Medical School in 1931, and pursued graduate training in obstetrics. He practiced that specialty and general medicine in Westfield until retirement in 1975. He had been affiliated with the Muhlenberg Hospital in Plainfield and with Rahway Memorial Hospital in Rahway.

Dr. Howard H. Israeloff

Notice has been received of the death on December 19, 1978, at Newark Beth Israel Medical Center, of Howard H. Israeloff, M.D., after a long illness. A native of Brooklyn, New York, Dr. Israeloff earned his doctorate of medicine from State University of New York Medical College in Brooklyn. He took graduate training in gynecology and was a Fellow of the International College of Surgeons. More recently he had practiced family medicine in Irvington and had been affiliated with the Beth Israel Medical Center and Doctors Hospital in Newark, and with Saint Barnabas Medical Center in Livingston. Dr. Israeloff was 71 years old at the time of his death.

Dr. Alphonse F. Norman

One of Middlesex County's senior members, Alphonse F. Norman, M.D., died on November 21, 1978. A native of Canada, born in 1902, Dr. Norman was graduated from Laval University Medical College in Quebec in 1932 and pursued graduate training in otorhinolaryngology, becoming board certified in those specialties. Now retired, he had practiced in Perth Amboy for many years and was on the staff at the Newark Eye and Ear Infirmary and the Perth Amboy General Hospital. He was a Fellow of the American College of Surgeons and of the American Academy of Ophthalmology and Otolaryngology. During World War II, Dr. Norman served in the medical department of the United States Air Force.

Dr. F.M. Offenkrantz

Frederick M. Offenkrantz, M.D., a member of our Union County component died in Mountainside Hospital, Montclair, on December 21, 1978. A native of New Jersey, born in 1913, Dr. Offenkrantz was graduated from Columbia University's College of Physicians and Surgeons, class of 1937, and took graduate work in pathology becoming board certified in clinical pathology. He was a Fellow of the American College of Pathology and had been associated with St. Michael's Medical Center in Newark.

Dr. Earl H. Snavely

Word has just been received of the death on July 2, 1978, of Earl H. Snavely, M.D., in Miami, Florida, where he was living in retirement. A native of Maryland, Dr. Snavely earned his medical degree from the University of Maryland School of Medicine in 1905 and pursued a career in psychiatry, becoming board certified in that specialty. He was a member of our Essex County component and a Fellow of the American Psychiatric Association and of the American College of Hospital Administrators, and was past president of the New Jersey Hospital Association. Dr. Snavely was 95 years old at the time of his death.

Dr. Willard H. Somers

A member of our Bergen County component, Willard H. Somers, M.D., died on February 18. A native of New York, Dr. Somers was graduated from Columbia University's College of Physicians and Surgeons in 1935. He pursued graduate work in obstetrics and gynecology, becoming board certified in those specialties. He was a Fellow of the American College of Surgeons and a member of the New Jersey Society of Obstetricians and Gynecologists. Dr. Somers was affiliated with the Englewood Hospital and the Bergen Pines Hospital in Paramus and had been chief of the department of obstetrics and gynecology at both facilities. Now retired he had practiced in Tenafly. During World War II, Dr. Somers served with the medical department of the AUS.

Dr. Henry O. vonDeilen

Henry O. vonDeilen, M.D., a member of our Morris County component, died on February 6. A native of Morristown, born in 1906, Dr. vonDeilen earned his medical degree from Columbia University's College of Physicians and Surgeons in 1932 and returned to his home town to establish a practice in obstetrics and gynecology, ultimately becoming chief of that department at Morristown Memorial Hospital. He was a Fellow of the American Academy of Obstetrics and Gynecology and a member of the New Jersey Society of Obstetrics and Gynecology. Dr. vonDeilen was active in medical society affairs and had been treasurer of his county society.

BOOK REVIEWS

Review of Medical Pharmacology

6th Edition. Meyers, Jawetz, and Goldfein. Los Altos, CA, Lange Medical Publications, 1978. pp. 762. Illustrated. ($14.50)

This book is written in the style of the other Lange publications. The authors' emphasis and approach to pharmacology serves the clinical needs of the student in medicine, dentistry, nursing, and pharmacy. Discussion of theoretical and research aspects of pharmacology is limited and in keeping with the review style.

The book has positive and negative aspects. Certain parts of the text are timely, including good discussions of cimetidine and the new non-steroidal, anti-inflammatory drugs. In addition, there are very useful tables in the appendix concerning the effects of a wide variety of drugs on common laboratory tests.

On the negative side, there is no discussion of the effect of aging on pharmacokinetics. The authors admit that the Preparations Available section has been hard to maintain accurately. A practicing physician, however, requires up-to-date information in this section. Pneumovax® was scarcely mentioned, but phenformin, removed from the pharmacist's shelf more than a year ago, received almost a complete page of discussion. The various anti-hypertensive agents are listed and their pharmacology reviewed, but the student is provided with little assistance in the step-wise approach to their use. In many aspects the text closely resembles a dictionary of drugs rather than a textbook.

In summary, medical and other health science students will appreciate the text for its conciseness and usefulness for examination preparation. However, the practicing physician, dentist, nurse, or pharmacist will have no use for it.

S.W. Warburton, M.D.

The Metabolic Management of the Critically Ill

(With support plan and work sheets) Douglas W. Wilmore. New York, Plenum, 1977. pp. 262. ($22.50)

This book discusses the various aspects of the problems that arise in treating the critically ill patient postoperatively and after serious injury. It is written in a question and answer form and thereby serves the purpose of being more informative without losing the attention of the reader, while allowing ready reference to specific areas of interest. The basic definitions are not forgotten and the reader is reminded of basic physiology which perhaps he has not reviewed recently or utilized. Adequate citation of reference material is included. A series of nomograms are presented together with their use in a *Support Plan* employing step-by-step management of various patient problems utilizing a metabolic and nutritional work sheet for recording patient data. The use of this type of analysis is of importance in patient management especially in those more complicated cases and where total parenteral nutrition is to be employed. Dr. Wilmore enhances the practical aspects of this book by bringing all of the nomograms together in a booklet and supplying individual patient work sheets (even with a red string for reading nomograms). Since this allows the practicing physician to apply basic physiological principles more simply to daily patient care, he is to be commended. This is useful, recommended reading for anyone who supervises the care of hospitalized patients.

Mark M. Singer, M.D.

Current Medical Diagnosis and Treatment.

Marcus A. Krupp, M.D. and Milton J. Chatton, M.D. Los Altos, California, Lange, 1979. Pp. 1130. ($18)

This 1979 revision is intended to serve, as did its successful predecessors, as a desk rather than a shelf reference for matters primarily related to internal medicine. It should serve this purpose well. The physician who is interested in pursuing topics to greater depths is provided with an excellent, updated bibliography. As a desk reference, *Current Medical Diagnosis and Treatment* contains a wealth of useful information, much of it in chart or tabular format, with occasional clarifying illustrations.

One is pleasantly surprised to find amidst the abundance of compressed data and descriptions some discussions, albeit brief, of subjects such as death and dying, psychiatric problems associated with medical and surgical disorders, genetic counseling, and medical recommendations for foreign travel.

It is recommended that a recent copy of this book be kept close at hand.

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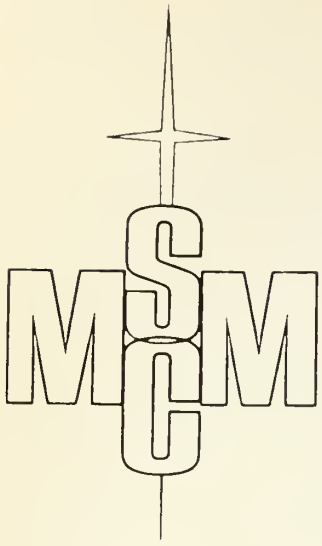
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Pelvic Ultrasound in Ectopic Pregnancy
M.A. Pelosi, M.D., et al.

Primary Carcinoma of Gallbladder
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Countercurrent Immunoelectrophoresis
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Medical Aspects of Middlebrook Encampments
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Precautions: Bronkodyl should be used with caution in patients with cardiac or circulatory disease.

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Dosage and Administration: *Adults:* Usual dosage of Bronkodyl is 200 mg. every 6 hours (four doses in each 24 hours). This dosage may be adjusted to reflect individual clinical response as an indication of slow or rapid metabolism of the drug. If adverse reactions are encountered, each dose may be reduced, or the interval between doses may be lengthened, or both. If clinical response is not satisfactory, indicating possible rapid inactivation of the drug, dosage may be gradually increased to achieve the desired response. In some instances of either too slow or too rapid metabolism, plasma levels of theophylline should be determined and dosage adjusted accordingly to achieve levels above 10 mcg./ml., but not to exceed 20 mcg./ml.

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The cover photograph of Saint Vladimir's Church, Cassville, New Jersey, taken in the Spring of 1978, was provided by John L. Krause, Jr., M.D., a member of our Camden County component, who has supplied material for several covers in the past, most recently for the January 1979 issue.

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Before prescribing, see complete prescribing information in SK&F Co. literature or *PDR*. A brief summary follows:

* **WARNING**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in postsympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone.

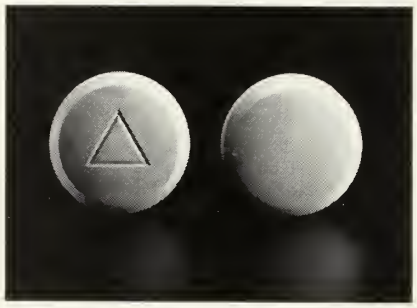
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The Maker

Examining a Few Myths About Prescribing.



Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.

MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

Matters.

MYTH: Generic options almost always exist.

FACT: About 55 percent of prescription drug expenditure is for single-source drugs. This means, of course, that for only 45 percent of such expenditure, is a generic prescribing option available.

MYTH: Generic prescriptions are filled with inexpensive generics, thus saving consumers large sums of money.

FACT: Market data show that you invariably prescribe—and pharmacists dispense—both brand and generically labeled products from known and trusted sources, in the best interest of patients. In most cases the patient receives a proven brand product. Savings from voluntary or mandated generic prescribing are grossly exaggerated.

MYTH: Drugs account for a major portion of the rise in health care costs.

FACT: Drugs represent a very small part of such costs. The amount of the health care dollar spent for prescription drugs was about 12 cents in 1967; today it is about 8 cents. And you as a physician are most conscious of how drug therapy can cut hospitalization, avert surgery, reduce office visits and keep patients on the job.

MYTH: Government intrusions into the marketplace will save tax money.

FACT: Government schemes always cost the taxpayer something, and the costs often exceed the benefits. Certainly, any federal "help," such as lists of wholesale drug prices sent to all physicians and pharmacists, will be no exception. Just think of the expense of keeping them current! Moreover, wholesale prices are poor guides to actual transaction prices and even worse guides to retail prices.

The PMA Position

We believe your freedom to prescribe, either by generic or brand name, should be totally unabridged. Otherwise, your prescribing prerogatives and your relationships with patients will be seriously impaired.

The maker does matter

After the myths about price and equivalency have been shattered, one fact stands out more clearly than ever: *The maker does matter.* As always, your best guide to drug therapy for your patients is to select products—both brands and generics—from manufacturers with credentials and performance records you have come to respect.

PMA

Pharmaceutical Manufacturers Association
1155 Fifteenth Street, N.W.
Washington, D.C. 20005

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Accident Facts. Chicago, Illinois, National Safety Council, 1974.

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Precautions: Use with caution in individuals with histories of significant allergies and/or asthma. Do not rely on oral administration in patients with severe illness, nausea, vomiting, gastric dilatation, cardiospasm, or intestinal hypermotility. Occasional patients will not absorb therapeutic amounts given orally. In streptococcal infections, treat until the organism is eliminated (minimum of ten days). With prolonged use, nonsusceptible organisms, including fungi, may overgrow; treat superinfection appropriately.

Adverse Reactions: Hypersensitivity, including fatal anaphylaxis. Nausea, vomiting, epigastric distress, diarrhea, and black, hairy tongue. Skin eruptions, urticaria, reactions resembling serum sickness (including chills, edema, arthralgia, prostration), laryngeal edema, fever, and eosinophilia. Infrequent hemolytic anemia, leukopenia, thrombocytopenia, neuropathy, and nephropathy, usually with high doses of parenteral penicillin. [102175]

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EDITORIALS

Health and Economic Principles

In a recent editorial in the *Trenton Times,* the writer started his discussion of hospital cost-containment by taking a swipe at President Carter's program which sets a "voluntary limit of 9.7 percent higher on hospital costs for the current year and requires mandatory controls in 1980 if that goal isn't met." He implied it is too weak. He ended with a blast at the "excessively high fees" that some doctors charge and culminated with the statement:

"There has been talk in Washington of uniform negotiated fees for doctors, instead of letting them set their own, but that's a long way down the road. A lot of pocketbooks could be bled white before that happens."

Economics was not one of the required pre-medical courses for any of us, but medical economics has become a prime consideration for all physicians in the past decade. The Federal Trade Commission (FTC) has entered the scene, along with the Department of Health, Education, and Welfare (HEW), and these two massive and powerful federal agencies seem bent on regulating the practice of medicine as government regulates the transportation industry, the utilities and communications industries, and those service-oriented industries which they deem to be "monopolistic."

In order to understand this situation at the simplest level, physicians need a scorecard and some definitions:

Demand and Supply: The ability and willingness of the consumer to spend his money on products and services are called *"demand."* Business and professions produce products and services. The total of all these goods and services available to the consumers at various prices is called the *"supply."*

Marketplace: The place where buyers and sellers come together to agree on the purchase of goods and services is the *"marketplace."*

Government Regulation: Through congressional acts, laws, and regulations, the government attempts to induce competition among companies in a given industry to assure a variety of goods and services at reasonable prices. Where a single industry is labeled a "public utility" (e.g., electric and gas companies), a monoply is preserved but prices are regulated by government.

Competition: When two or more companies offer similar products or services to consumers, they are said to be in *"competition."* Competing firms attempt to provide the best product or service at a price high enough to assure a profit yet low enough to attract a share of the market. Monopoly exists in the absence of competition.

How does all this relate to the practice of medicine? Is there a "medical marketplace?" Is medicine a "monopoly," i.e., is there no competition in the delivery of health services? Do the laws of "demand and supply" apply to the health industry? Should the health industry be treated like a public utility?

The answers to these questions are easy if you work for FTC or HEW—*Yes* to all! But, will the application of these economic principles preserve a health system which will provide high-quality services in adequate quantities to satisfy the demands of the consumers at controllable costs? Will the providers and the consumers be satisfied with the end result? Only a crystal ball can answer these questions.

The question of "demand and supply" would not appear to be relevant because the American public seems to have an insatiable appetite for health services. Despite advice to the contrary, patients continue to demand expensive drugs and treatments for acute respiratory diseases which do not require treatment; they insist on instant "cures" for ailments that have a natural course to run; and, they require removal of innocent skin lesions for fear they may become cancerous. The litigious patient and some members of the legal profession have forced the medical profession into a mindless defensive practice approach (which is extremely costly) in order to avoid overlooking all possibilities, no matter how remote. Emergency room services skyrocket each year and billions of dollars are spent for over-the-counter drugs.

There is alleged to be an oversupply of surgeons in some communities, a shortage of primary physicians in others, and a "maldistribution" in general. Yet, surveys have shown that most patients have a family doctor and believe that health care by their physician is good to excellent, and that the fees which they pay the physician are reasonable.[1] There is no evidence that the presence or absence of competing specialists or primary physicians in a community determines fees and one can be certain that "advertising" fees and services by some physicians will not influence the behavior of the medical community as a whole.

At present in New Jersey there do indeed exist competing "systems." There are private, fee-for-service physicians in office and hospital practice who do not accept third-party payment. There are physicians who accept Medicare, Medicaid, or other insurance payments as full payment for office and hospital services. There are alleged to be "Medicaid mills." There are free-standing health maintenance organizations of the closed-panel type and individual practice organizations of the IPA type. There are hospital clinics, insurance-company sponsored industrial clinics, union health centers, veterans' administration outpatient facilities, and neighborhood family health centers. There are adequate numbers of physicians and centers who supply secondary and tertiary services with various reimbursement mechanisms. No physician or group practice or hospital can claim to have a monoply of any health service in our state. If this

is a fact, then there does exist a "functional marketplace" for health services.

Some take the view that the health industry is "monopolistic" or "imperfectly competitive" because "consumers are not well aware of price, attractiveness, and quality for the many alternative sources of medical care."[2] Medicine and surgery are complex and growing more so. There is almost no way that advertising or general health education can inform patients widely and deeply enough so as to make wise choices in every instance. Can the patient in pulmonary edema gasp "help me!" or "what is your fee and what are your qualifications?" Can the cardiologist treating ventricular fibrillation or the surgeon advising an operation for rectal carcinoma "sell himself" to the patient? Even the second and third opinion concept cannot solve this dilemma.

A major problem in the concept of competition in health services is the fact that there is no incentive for the patient to search or concern himself with costs because health insurance pays all or most of the fee. If it came out of his own pocket, he well might take as large an interest as he does when he purchases a home appliance or automobile. Neither complete payment by an insurance company nor by the government through national health insurance, with or without a coinsurance feature, will reduce the demand for medical care.

Do some physicians and surgeons take advantage of the system and charge fees which are excessive? This is obviously the case as is attested to by the work of county medical society judicial committees. However, the numbers of complaints are not large, while the peer-review concept grows increasingly powerful and effective where inequities do exist.

Government regulation of the utility industries does not appear to control costs. Cost control in the communications industry has been largely the result of scientific advancement rather than government regulation. Why should the American public believe that government will control health costs through regulation when the experts in the field cannot figure out how to control inflation? Kenneth Galbraith believes the only cure for inflation pathology is "permanent government controls of prices and wage rates."[3] That does not apply only to the health industry, but to the wages of congressmen, union leaders, lawyers, truck drivers, baseball professionals, and every other wage earner in the United States!

When this country is ready to accept that national policy —and politicians have the courage to run for election on it— then physicians will have no choice but to let others set their fees. Until then, the arguments to control physicians' income run thin.

Medical economics is no less complex than general economics, and for intrinsic reasons, may be more so. The American health industry cannot be stuffed into an economic model based on supply—demand—competitition—monopoly—marketplace—regulation. If the politicians do that, the system simply will not conform to the democratic concepts on which this nation was founded and the American public will receive second-rate care. A.K.

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[2]Frech HE and Ginsburg PB: *Public Insurance in Private Medical Markets*. Washington, DC, American Enterprise Institute for Public Policy Research, 1978, p. 20.

[3]Samuelson, PA: The roots of inflation. *Britannica Book of the Year*. Chicago, Encyclopaedia Britannica, 1979, p. 64.

Our New Headquarters*

The Medical Society of New Jersey officially has taken up residence in Lawrenceville, New Jersey. Although far from completed, the interior has begun to shape up as a spacious and efficient working and meeting environment. Its beauty will come as finishing touches slowly are applied to the interior and exterior.

Meanwhile, with the first meeting of the Board of Trustees on Sunday, February 25, 1979, its occupancy became a reality. Since councils and committees regularly will be using its space, some directions are in order:

Northern Counties: Take your best route to New Jersey Turnpike South thence to Interchange #9 (New Brunswick). Exit Turnpike at #9 and take State Route #18 West to U.S. Route #1 South. Follow Route #1 South to Interstate #295. Follow signs for Interstate #95 South and 295 (Pennsylvania). Exit 295 at Princeton Pike (Exit 68B). At top of exit ramp, turn right onto Princeton Pike, crossing over Interstate 295, to Princess Road, first road on left, turn left.

Northwestern Counties and Pennsylvania: Take your best route to Interstate #95 North. Follow Interstate #95 to where, due to incomplete construction, Interstate #95 becomes Interstate #295 South. Follow Interstate #295 and use Exit 68B. At bottom of egress ramp, you will be on Franklin Corner Road (County Rt. #546 East). Proceed to next intersection, Princeton Pike. Turn left onto Princeton Pike (County Rt. #583 North). At the next road on right, Princess Road, turn right.

Eastern Counties: Take your best route to Jackson, New Jersey. Enter Interstate #195 and follow to end of roadway (Arena Drive). Turn right onto Arena Drive and follow signs to Interstate #295 North entrance. Follow Interstate #295 to Princeton Pike (Exit 68B). At top of exit ramp, turn right onto Princeton Pike, crossing over Interstate #295, to Princess Road, first road on left, turn left.

Southern Counties: Take your best route to either New Jersey Turnpike North or Interstate #295 North.

If Turnpike North, exit at Interchange #7 (Trenton).

If Interstate #295 North, exit at "McGuire AFB—Fort Dix" Exit 55 Follow signs to U.S. #206 North. Follow U.S. #206 to Junction with U.S. Route #130 North. Follow signs for U.S. #130 North. Ignore signs for Interstate #295 North, staying on U.S. #130 North for three miles to Klockner Road. (Landmarks—Harry's Sporting Goods on right—U.S. Postal Distribution Center on left). Turn left onto Klockner Road following it for approximately four miles to Hamilton Avenue. Turn right onto Hamilton Avenue and follow to entrance of Interstate #295 North. Follow Interstate #295 North to Exit 68B, Princeton Pike. At top of exit ramp, turn right onto Princeton Pike, crossing over Interstate #295, to Princess Road, first road on left, turn left.

Ultimately, the House of Delegates will be able to meet in the auditorium. Meanwhile, it's *your home*, so do plan to visit when you can. A.K.

*See area map, page 392, this issue.

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April 1979



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Pelvic Ultrasound: A New Aid in the Diagnosis of Ectopic Pregnancy

M.A. PELOSI, M.D., Newark
P.J. GOLDSTEIN, M.D. and
R.J. D'AMICO, M.D., Bayonne*

Recently, ultrasonography has become an important diagnostic procedure in obstetrics and gynecology. This article illustrates the value of that relatively new diagnostic aid in the detection of early ectopic pregnancy. The ultrasonic signs and criteria for diagnosis of ectopic pregnancy are outlined and illustrative pelvic sonograms of patients with early ectopic pregnancy are presented. The technique is simple, noninvasive, and rapid. It should be a standard procedure in the evaluation of patients with suspected ectopic pregnancy.

Approximately five to seven percent of maternal deaths in the United States are due to complications related to extrauterine gestations.[1] When a patient presents the classic clinical picture of normal menstruation replaced by vaginal bleeding, pelvic pain, and the finding of a tender adnexal mass, accompanied or not by signs of hemorrhagic shock and peripheral collapse, there is little difficulty as to the diagnosis. Unfortunately, the clinical manifestations of ectopic pregnancy before rupture or abortion are not characteristic.

The early and rapid detection of ectopic pregnancy reduces potential complications and simplifies its management. Because of the difficulties in early recognition, a variety of procedures have been utilized to increase diagnostic accuracy. These include serial hematocrit determinations, pregnancy tests, culdocentesis, colpotomy, dilatation and curettage for evaluation of the endometrium, culdoscopy, laparoscopy, and hysterosalpingography.[1-3] Unfortunately, false negative as well as false positive diagnoses are not unusual, although the error is less than fifteen percent with the operative methods.[4]

It is the purpose of this article to illustrate the value of the relatively new diagnostic aid in the diagnosis of early ectopic pregnancy. We consider that pelvic ultrasonography should be established as standard procedure in the modern evaluation of patients with suspected extrauterine pregnancy.

BACKGROUND

Ultrasonography, a noninvasive technique, recently has become an important diagnostic tool in obstetrics and gynecology. Ultrasound is valuable in determining the presence of pregnancy, establishing gestational age, delineating the position, size, and characteristics of the placenta in the diagnosis of a blighted pregnancy, and for evaluation of fetal growth and development. Fetal structures, other than the biparietal diameter of the head, often can be demonstrated and various anomalies can be diagnosed prenatally. It is useful in the diagnosis of fetal death. Demonstration of the position and orientation of the fetus with easy recognition of cephalic presentation, breech presentation, or transverse lie can be made. It is useful in the detection of multiple pregnancies and of abruptio placenta. It is also used for determining the quantity of amniotic fluid. The appearance of trophoblastic disease on ultrasound examination is pathognomonic. Ultrasound is also helpful in evaluating the pelvic region for uterine and extrauterine masses that may compromise the passage of the fetus through the birth canal. It has proved to be an effective method of needle localization in amniocentesis.

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Presently, ultrasound is the imaging method of choice for evaluating the pregnant abdomen. In addition to its obstetrical uses it is a very important noninvasive tool in the diagnosis and management of gynecologic disease. The normal uterus and, less frequently, the normal ovaries can be imaged. Masses originating in these organs can be demonstrated when physical examination is inconclusive. The characteristics of these masses, whether they are solid, cystic, or complex, can be determined accurately. Ultrasound also has proved useful in evaluating patients with ambiguous genitalia for determining the presence and size of the uterus. Evaluation of the amenorrheic patient is enhanced by ultrasonography. Hematometrium and hematocolpos are demonstrable. Congenital variations, such as a double or bicornuate uterus, can be imaged. It is useful in patients with suspected pelvic inflammatory disease and is also an effective means of localizing intrauterine contraceptive devices and foreign bodies.[5]

Patient preparation for ultrasound studies is minimal. It is desirable to have the patient with a distended urinary bladder. The scanning procedure is done with the patient lying supine. In general, scans are performed in both longitudinal and transverse planes. The longitudinal scans are important to demonstrate the uterus and to outline adnexal masses. The transverse scans are helpful in separating the uterus from adnexal masses and fluid in the cul-de-sac.

Normally, the nongravid uterus does not show intrauterine echoes even during menstruation, although occasionally an even low amplitude echo "fill in" may be seen. A distended bladder helps to elevate the uterus and to make the entire uterine outline visible. The vagina appears as a double line in the longitudinal scans. The cervix, if needed, can be outlined. Normal ovaries and tubes are not visualized routinely because of their relatively small size and their position behind the bowel. Under favorable circumstances, the normal ovaries can be seen as very weakly echogenic ovoid masses two to three cm in size.

Ultrasound is capable of detecting evidence of pregnancy as early as five weeks of gestation with the demonstration of the developing gestational sac. (Figure 1) Gestational sacs of one cm or greater in diameter can be detected. At approximately eight weeks, a cluster of echoes usually is detected eccentrically within the gestational sac that represents the earliest fetal development. As the gestational sac develops, a thickening of one wall, which represents early placental development, is seen. The sac continues to enlarge and persists until the tenth week. At the 10th to 12th week of pregnancy, the gestational sac becomes filled with fetal structure; at that time it may be difficult accurately to assess the age of pregnancy. The fetal head is readily detected after the 12th week of pregnancy.[4-6]

Despite the extensive literature related to ultrasound as a diagnostic aid in obstetrics and gynecology, its use as a nonoperative diagnostic procedure in the evaluation of patients with ectopic pregnancy and its diagnostic accuracy rarely have been reported. Over 90 percent of ectopic pregnancies occur in the fallopian tube. About 60 percent of these are located in the ampullar portion or fimbriated end, about 25 percent in the isthmic portion, and five percent in the interstitial part. The remainder are rare forms of intraligamentous, ovarian, abdominal, or cervical pregnancies.

In 1969, Kobayashi *et al.* described the appearances of ectopic pregnancy utilizing bistable B-scan ultrasonography.[4] Their overall diagnostic error was 25.6 percent. The major diagnostic criteria were uterine enlargement with diffuse amorphous intrauterine echoes with absence of intrauterine pregnancy. An irregular, fairly diffuse extrauterine mass was a contributory diagnostic aid. They recognized the limitations of the equipment used at that time and correctly predicted that standardization of techniques and better equipment will improve the diagnostic accuracy. Maklad and Wright recently have reported the gray scale ultrasonographic appearances of ectopic pregnancy and reviewed the literature on the subject.[6] Their study included 36 patients with suspected ectopic and three with pelvic mass and bleeding. The overall diagnostic accuracy was 92.3 percent. There were one false-positive and two false-negative studies. The major diagnostic criteria in unruptured ectopic pregnancy were an extrauterine gestational sac or fetus and an abnormal uterine echo pattern. (Figure 2) In addition a complex mass due to hematoma was seen in ruptured cases. These authors have outlined the utrasonic signs and criteria for diagnosis of ectopic pregnancy.

In unruptured ectopic pregnancy, the ultrasonic signs are:

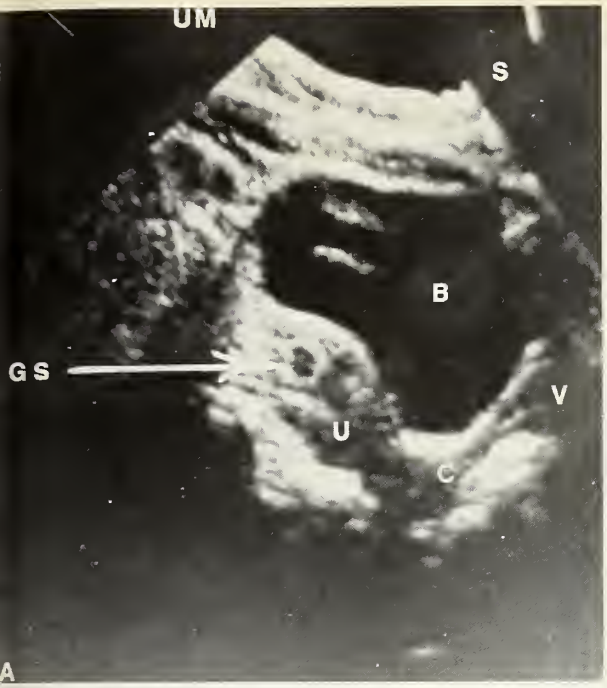
1. Visualization of a gestational sac or fetus in an extrauterine location.
2. An oval or elongated fluid-filled adnexal mass (distended fallopian tube) containing an echodense ring-like structure representing the gestational sac.
3. A moderately enlarged uterus, with no intrauterine gestation, but with an abnormal uterine echo pattern, giving the uterus a mottled appearance.
4. Detection of fetal heart motion within the extrauterine mass, utilizing a real time scanner. This sign is only present if the fetus is viable and the duration of pregnancy is at least ten weeks.

In **ruptured ectopic pregnancy** the ultrasonic signs are:

1. Extrauterine gestational sac in association with a complex mass representing hematoma.
2. Displacement or deviation of the uterus by an oval complex mass, and free fluid in the cul-de-sac.
3. A moderately enlarged uterus, with no intrauterine gestation, with or without the abnormal echo pattern.

The abnormal uterine pattern is possible due to increased vascularity of the myometrium, decidual reaction, and endometrial shedding. Ultrasonic examination showing the absence of intrauterine pregnancy is only presumptive evidence of ectopic pregnancy. When pregnancy is clearly identified within the uterus, it is unlikely that ectopic pregnancy coexists. However, Perlmutter and Goldberg[5] illustrated a case of a simultaneous intrauterine pregnancy and a right tubal gestation diagnosed by ultrasound. Chronic ectopic pregnancy remains the most difficult to diagnose and usually simulates pelvic inflammatory disease or inflammatory masses. The only consistent finding is displacement or deviation of a slightly enlarged uterus by a complex mass.

The following are illustrative pelvic sonograms of patients with ectopic pregnancies. Figures 1, 2, 3, 4:



igure 1 (A)—Normal intrauterine pregnancy. Longitudinal scan. rrow indicates a six-week gestational sac (GS). The uterus (U) lies osterior to the bladder (B). Vagina (V). Uterine cervix (C). Sym-hysis (S). Umbilicus (UM).

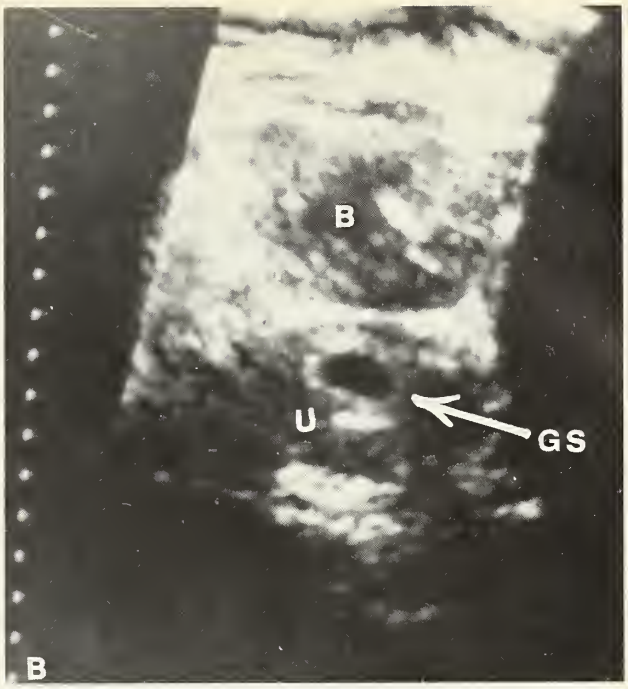
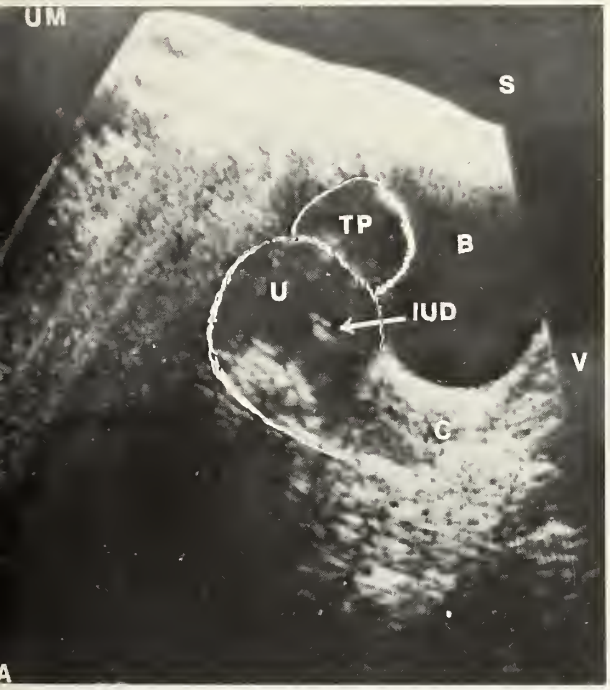


Figure 1 (B)—Transverse scan from the same case. Arrow indicates the gestational sac (GS). Uterus (U). Bladder (B).



igure 2 (A)—Unruptured left tubal pregnancy. Longitudinal scan. left unruptured tubal pregnancy is represented by an adnexal mass hich contains complex echoes (TP). The uterus (U) is enlarged and es posterior to the bladder (B). An intrauterine device (IUD) is seen the uterine cavity. No intrauterine gestational sac is present. Uterine cervix (C). Vagina (V). Symphysis (S). Umbilicus (UM).

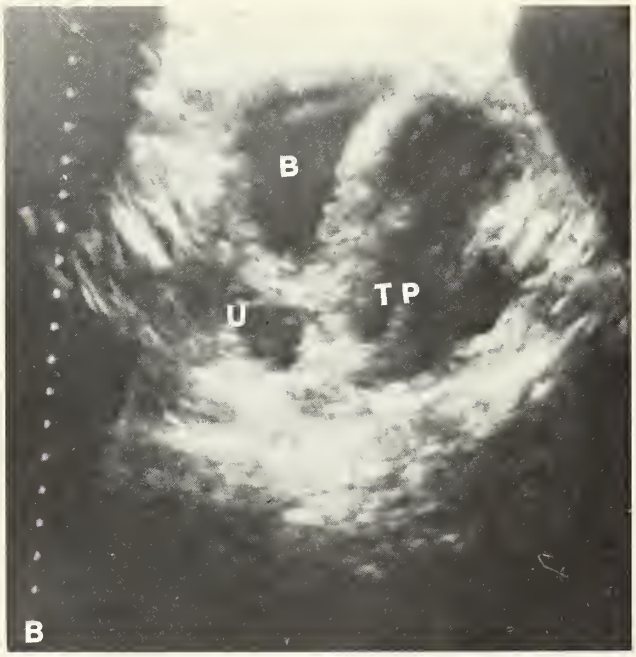


Figure 2 (B)—Transverse scan from the same case. The tubal pregnancy (TP) is represented by the left adnexal mass. The empty uterus (U) is deviated to the right by the extrauterine gestation. Urinary bladder (B).

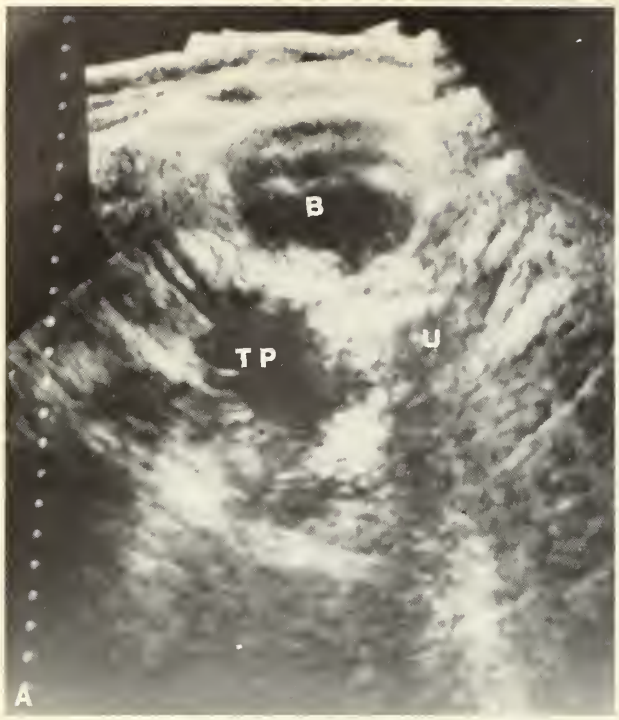


Figure 3 (A)—Unruptured right tubal pregnancy. Transverse scan. The tubal pregnancy is represented by a right adnexal mass which contains irregular echoes (TP). The empty uterus (U) is displaced to the left by the mass. Urinary bladder (B)

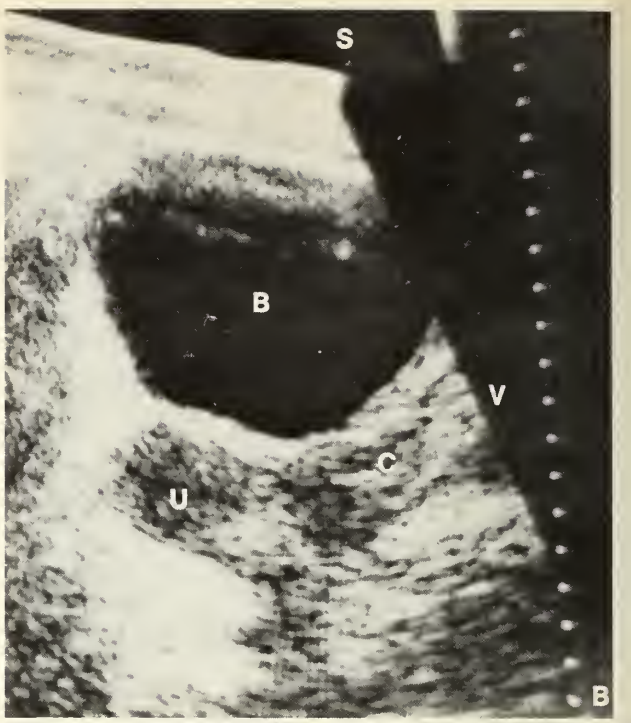


Figure 3 (B)—Longitudinal scan from the same case. The sonogram reveals a slightly enlarged uterus (U). No gestational sac is identified in the uterine cavity and there is an abnormal uterine echo pattern giving the uterus a mottled appearance (decidual reaction). Urinary bladder (B). Uterine cervix (C). Vagina (V). Symphysis (S).

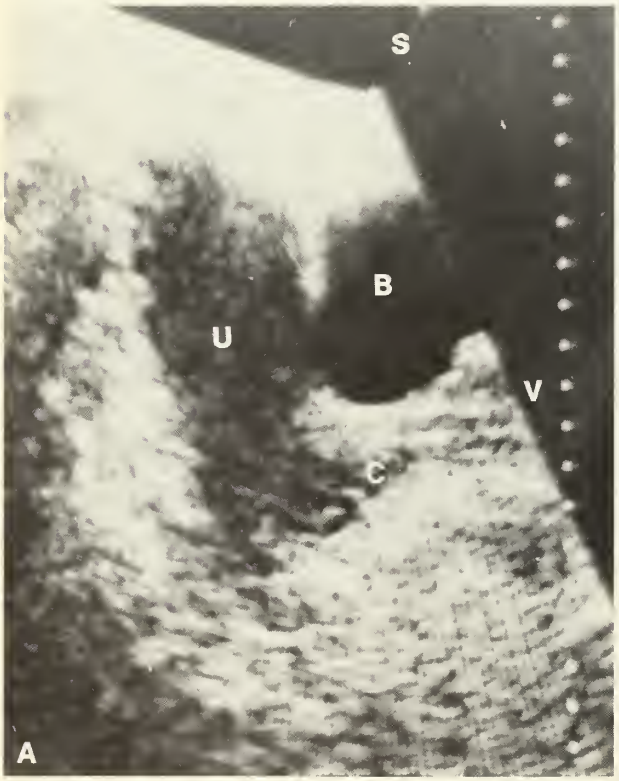


Figure 4 (A)—Unruptured left tubal pregnancy. Longitudinal scan. Moderately enlarged uterus (U) lies posterior to the urinary bladder (B). No intrauterine gestational sac is present. An abnormal intrauterine echo pattern is present (decidual reaction). Uterine cervix (C). Vagina (V). Symphysis (S).

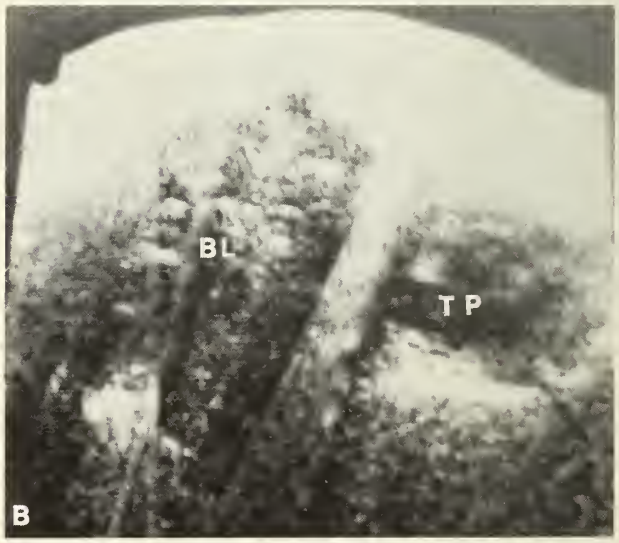


Figure 4 (B)—Transverse scan from the same patient. The tubal pregnancy (TP) is represented by a left adnexal mass which contains a complex echo. The uterus is not visualized because this view is high above the symphysis pubis. Urinary bladder (BL).

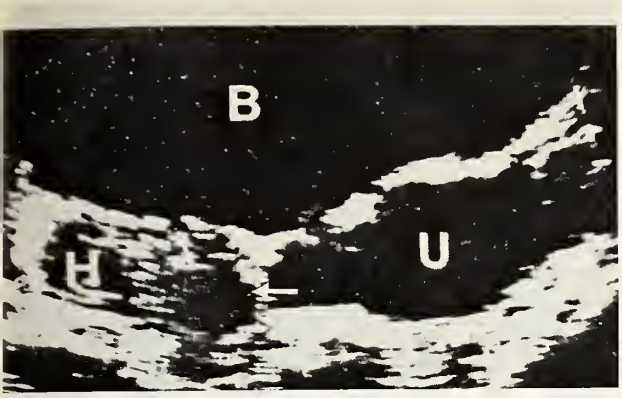


Figure 5—Ruptured ectopic pregnancy. Transverse scan. The gestational sac (arrow) lies to the right of the uterus (U) and is associated with a complex mass (H) representing a hematoma. Bladder (B). From Maklad, NF and Wright, CH: Gray scale ultrasonography in the diagnosis of ectopic pregnancy. *Radiology* 126:221, 1978

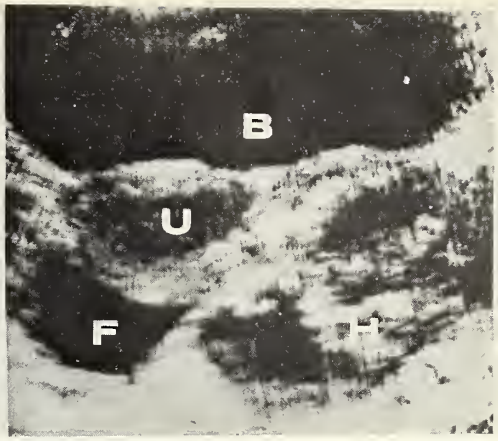


Figure 6—Chronic ruptured ectopic pregnancy. Transverse scan. The uterus (U) is displaced by a large complex mass in the left adnexa representing a hematoma (H), and by free fluid in the cul-de-sac (F). From Maklad, NF and Wright, CH: Gray scale ultrasonography in the diagnosis of ectopic pregnancy. Radiology 126:221, 1978

Two additional ultrasonograms are included here. One corresponds to a ruptured ectopic pregnancy (Figure 5) and the other to a chronic ruptured ectopic pregnancy. (Figure 6)[6]

COMMENT

The clinical diagnosis of early ectopic pregnancy can be very difficult. The risk to the patient is particularly serious if a false-negative diagnosis delays surgical exploration. In order to increase diagnostic accuracy ancillary diagnostic procedures usually are necessary. Nevertheless, at the present time laparotomy should not be the inital procedure of choice in the diagnosis and management of gynecologic disease. With some exceptions a more conservative approach can be instituted prior to exposing the patient to operative and anesthetic risks. Pelvic ultrasound has become a very useful tool in the diagnosis of gynecologic conditions, and its value in the evaluation of patients with suspected early ectopic pregnancy clearly is appreciated.

Pelvic ultrasonography should be performed on any patient with suspected extrauterine gestation. In that way, this potentially lethal condition can be diagnosed at an early stage, resulting in decreased morbidity and mortality. The technique is simple, noninvasive, rapid, and should be incorporated as standard procedure in the modern evaluation of patients with suspected ectopic pregnancy.

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Primary Carcinoma of the Gallbladder

PAUL J. MARSIDI, M.D.,
MUHAMMAD ANWAR, M.D., and
GEORGE O. HALSTED, M.D., Englewood

Primary carcinoma of the gallbladder is relatively infrequent, so the individual surgeon has a limited experience in the treatment of this disease. Presenting symptoms are usually similar to benign disorders of the gallbladder, therefore the diagnosis is made too late and limited surgical treatment usually is inadequate. Thirty-one patients who were seen in a ten-year period at Englewood Hospital are presented. Signs and symptoms, pathology, length of survival, and treatment are discussed with emphasis on the role of surgery.

Although primary carcinoma of the gallbladder is uncommon, the disease is not rare and continues to be a diagnostic and therapeutic challenge. Carcinoma of the gallbladder presents either as an unresectable tumor discovered at exploration for obstructive jaundice. The survival rate is extremely poor and few advances have been made in the treatment of this disease.

It is the purpose of this article to review critically our experience with this highly malignant disease in an attempt to determine the reason for the poor results and to discover the best surgical management. This report summarizes the course of patients with carcinoma of the gallbladder seen at Englewood Hospital in a ten-year period.

METHODS

We reviewed the courses of thirty-one patients with carcinoma of the gallbladder from 1965 to 1975 at Englewood Hospital in Englewood, New Jersey. The charts were studied to obtain the following data: age, sex, signs and symptoms, pathology, treatment, length of survival, and results. All the diagnoses were made by pathological examination.

Age and Sex Distribution—The ages of our patients at the time of diagnosis ranged from 36 to 84 years, the average being 70 years (Figure 1). Twenty-five of the patients were women and six were men, a sex ratio 4:1 which is in general agreement with the figures in the literature. [1,2,3,4,5]

Signs and Symptoms (Table 1)—The most common presenting symptom was abdominal pain which ranged from a vague epigastric fullness to severe colicky pain. Fifteen patients complained of pain localized to the right upper quadrant. In two patients, the pain was localized in the epigastrium; two patients complained of difficulty in breathing and one had pleural effusion on the right side. The duration of the symptoms ranged from one day to six to eight weeks.

In eleven patients, mild to severe jaundice was noted; some complained of pruritus and change of color of stool and urine. The duration of the jaundice was one week in most of the cases. Eight patients were admitted with a history of weight loss and poor appetite of two weeks to six months duration. Nausea and vomiting were reported by nine patients.

Studies—Cholecystography was performed in nineteen patients, but was not helpful in making a correct diagnosis of carcinoma of the gallbladder. In the majority of the patients, the gallbladder was not visualized and only stones were noted. In one patient, a complete obstruction of the right

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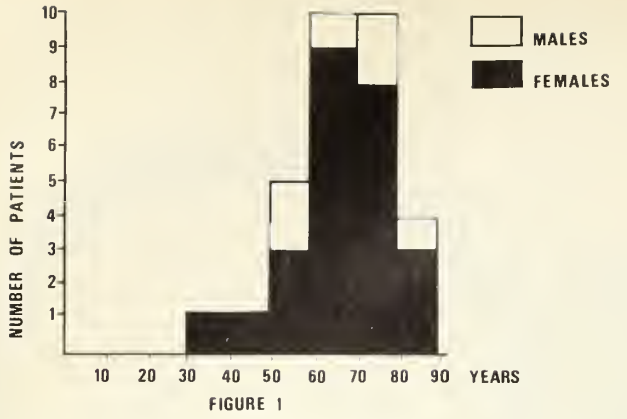


FIGURE 1

Figure 1—Age and sex distribution of patients with cancer of the gallbladder.

hepatic duct was noted during an operative cholangiogram; two patients had a dilated common duct.

Other roentgenologic studies also were not very helpful. In one case, an extrinsic mass on the duodenal bulb was noted with the possible diagnosis of gallbladder carcinoma.

Laboratory investigation revealed elevated alkaline phosphatase in twelve patients. Hyperbilirubinemia was noted in eleven patients. Liver profile and routine laboratory tests were usually within normal limits.

Treatment—Of the thirty-one patients who underwent operations, twenty had a simple cholecystectomy; three patients were given chemotherapy in the form of 5-fluorouracil. One patient received radiotherapy after cholecystectomy. One patient underwent right hepatic lobectomy, while another had a cholecystectomy. A right transverse colon resection along with cholecystectomy was performed on one patient. The liver was biopsied in ten patients. Peritoneal, retroperitoneal, and omental biopsies were done in eight patients and a gallbladder biopsy was done in one patient.

Pathology—Histologic examination of the tumor revealed a well-differentiated adenocarcinoma in twenty of the patients, papillary adenocarcinoma in four, differentiated carcinoma in two, and squamous cell carcinoma in four.

No specific histologic type was associated with long-term survival. The long-term survivors were those patients who had a well-differentiated adenocarcinoma. The most commonly involved organs were the liver, lymph nodes, omentum, peritoneum, and small and large intestines.

To estimate the frequency of gallstones among gallbladder cancer patients, only those patients whose gallbladder had been pathologically examined post-operatively or at necropsy, were taken into account. Eighteen of the twenty patients (90 percent) who underwent cholecystectomy had associated cholelithiasis; this incidence is the same as described by Littwin.[6] In thirteen patients, acute hemorrhagic or chronic cholecystitis was found.

Survival—Follow-up studies were done on all patients (Table 2). Twenty-one patients (67.7 percent) who were operated on died within one year after the operation. A similar incidence was reported by Ram.[7] One patient survived a period of fifteen months and one died twenty-nine months after surgery. Two patients who received chemotherapy survived one and three months, respectively.

Eight patients are still living, four of them ten months, sixteen months, forty-six months and three years, respectively and four patients have survived more than five years. All

Table 1
Signs and Symptoms

Findings	*No. of Patients (31)*	*Percent of Sample*
Abdominal pain	17	54.8
Jaundice	11	35.5
Weight loss	8	25.8
Nausea and vomiting	9	29.0
Right upper quadrant mass (palpable)	7	22.6

Table 2
Length of Survival of the Patients Who Died of CA of the Gall bladder

Length of Survival	*No. of Patients*	*Percent of Sample*
1-30 days	6	19.4
1-6 months	11	35.5
7-12 months	4	12.9
13-24 months	1	3.2
25-60 months	1	3.2

of the surviving patients underwent cholecystectomy as a curative procedure, one patient received chemotherapy post operatively. Seven patients had adenocarcinoma and one of these eight survivors had a papillary adenocarcinoma.

DISCUSSION

The prognosis of carcinoma of the gallbladder is poor because the diagnosis is made too late. Since the clinical features of the disease are so similar to benign disorders of the gallbladder, the diagnosis is seldom made preoperatively.[5] When it becomes more symptomatic, suggestive of malignancy, it has become inoperable.

Most of the patients underwent cholecystectomy as the primary operative procedure for gallbladder carcinoma. Nevertheless, most patients die of the disease with great rapidity as is shown in our series. The reason for this rapid death remains unexplained; it does not appear to correspond with the extent of the disease.

Pemberton found broncho-pneumonia and pulmonary embolus due to dehydration, malnutrition, and immobility as the cause of death in 90 percent of the cases.[8]

Chemotherapy as sole or adjuvant treatment has little effect on inoperable gallbladder cancer. Davis, *et al.* found a median longevity of four months after systemic or local intra-arterial infusion of 5-FU.[9] In our series, no patient who had liver involvement survived over one year. Cholecystectomy alone as a curative procedure has failed to improve survival because it overlooks any local or lymphatic spread. Fahim advocates a right hepatic lobectomy, since cholecystectomy with wedge resection alone produced only 5.9 percent five-year cure. This procedure may eradicate the primary and hepatic involvement in early cases, but it overlooks other possible sites of spread, especially the lymph nodes. To be adequate, the operation for early carcinoma of the gallbladder should consist of a cholecystectomy, wedge resection of the liver with a margin of free tissue, and adequate nodal dissection. If the liver is involved, the operation should consist of a cholecystectomy, right hepatic lobectomy, or a radical hepatic lobectomy which includes the right lobe as well as the medial segment of the left lobe of the liver (extended right hepatic lobectomy), adequate node dissection including the pericholedochal nodes and the celiac nodes, and resection of neighboring involved organs.

Adson believes that prophylactic cholecystectomy clearly is indicated for patients with symptomatic stones and ad-

vised surgical treatment of asymptomatic gallstones when minimal operative risk can be anticipated.[5] This refers to the patient who is less than 65 years of age and has no concomitant disease. By doing this, the risk of possible malignant transformation at a later date is avoided. The fact that asymptomatic gallstones can become symptomatic also is being considered when one recommends a prophylactic cholecystectomy.

SUMMARY

The surgeon's role in the treatment of primary carcinoma of the gallbladder is limited. Early diagnosis is seldom possible and surgical prophylaxis often is impractical. Seventy-five percent of the patients have unresectable disease at the time of surgical evaluation. For the others, the lesion is most often an incidental finding during surgery for calculus disease.

The association of gallbladder cancer and cholelithiasis is well established.[5] Gallstones were present in 90 percent of the pathological specimens of our patients. However, the etiologic relationship between gallbladder carcinoma and gallstone remains unclear. Cholesterol and bile salts, which are chemically related to specific carcinogens like methylcholantrene, might act as an endogenous carcinogen. Although the exact relationship is yet unknown, elective excision of diseased gallbladder would reduce the later development of carcinoma.[3]

Knowing that some of the early carcinomas are incidental findings, the gallbladder always should be opened during cholecystectomy and a frozen section should be taken of any suspicious lesion.[1,5]

If carcinoma is found, a more aggressive surgical approach must be taken and the above-mentioned operative guidelines of Fahim should be followed.[10] Histologic study of the margins of resected tissues, including the liver bed and cystic duct is relevant to proper therapy. Cholangiographic studies for evaluation of multicentric tumor involving biliary ducts is necessary.

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Simplified Deep Venography of the Legs

JONATHAN ADLER, M.D., Englewood*

A rapid procedure of peripheral venography is advocated to diagnose phlebitis and identify patients at high risk of pulmonary emboli. The procedure is useful in debilitated, elderly, paralyzed, or comatose patients, as well as ambulatory patients requiring a treatment decision. The radiographic findings reflect the time progression of the disease process within the veins. The examination aids the clinician to decide confidently whether or not to use heparin, or to choose a surgical technique to prevent embolization

The clinical signs and symptoms of lower extremity phlebitis may confuse experienced examiners so that many patients have been placed on heparin anticoagulation with only a presumptive diagnosis. The potential complications of anticoagulation stimulated an attempt to prove the diagnosis of phlebitis prior to treatment with heparin.

That there are several published techniques of venography indicates dissatisfaction and frustration among angiographers performing the procedure. Interpretation of the films is difficult due to anatomic variation and technical artifacts during each examination.[1,6-9,11,14]

A simple, rapid procedure of peripheral venography in patients suspected of having phlebitis and risk of pulmonary emboli was developed by a modification of existing techniques. Filling defects or occlusions in major veins readily can be observed with minimal discomfort to the patient. Patterns were noted in several serial studies to document the progression of the disease and evaluate the effects of the heparin therapy with recanalization of previously occluded veins.

METHOD

We have performed venography using our technique in over three hundred examinations in patients of both sexes and all ages suspected of having phlebitis during the years 1973 to 1978 at three institutions.

The patient is placed supine on any radiographic table. Heated towels are applied to the dorsum of the feet for five to ten minutes and a tourniquet is applied above the ankle to dilate the veins. Venipuncture is performed with insertion of a 23 or 21-gauge scalp vein butterfly needle and blood is permitted to fill the tubing before connecting D5/W or normal saline infusion drip. The needle is secured gently with tape and the puncture site closely inspected for infiltration with the tourniquet off. Fifty cc of Renografin 60® is injected at one to two cc/sec. by hand or with an automatic injector. The tourniquet is applied tightly above the ankle to force contrast medium into the deep veins. The venipuncture site is observed closely during the injection. There is immediate interruption of the injection if extravasation occurs.

Filming requires the technical assistance of two persons in addition to the person performing the injections in order to move the patient and change the cassettes rapidly. Four or five 14x17 films are taken in the following sequence:
After half the volume of contrast is injected (25 cc.)
(1) *Leg*—ankle to knee, AP
(2) *Knee*—include distal femur, AP
(3) *Hip*—include proximal femur, AP

These films are taken as quickly as the patient can be positioned with completion of the remaining 25 cc. of

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Figure 1—Filling defects in multiple deep veins represent acute clots which have formed within one week of the examination.



Figure 2—Filling defects propagate and obstruct normal flow patterns, encouraging collaterals in muscular branches.

contrast by the time the third film is exposed. After the tourniquet is released one or two more films are taken.

(4) *Knee*—AP

(5) *Knee and Proximal Tibia*—lateral (optional)

After filming, the intravenous fluid is run quickly to wash out contrast and the patient's feet are exercised.

RESULTS

Normal Venograms—

Normal venograms showed good opacification of deep leg veins, popliteal, and superficial femoral veins with no filling defects, distortion of valves, irregular vein walls, or occlusions. Many of this group were out-patients, largely young females, some with and others without a history of taking birth control pills; they presented with exquisite tenderness of the calf, swelling, and pain. The injection of contrast media usually was painless. Prior to this study, many of these patients were started on heparin therapy with the presumptive diagnosis of thrombophlebitis. A negative venogram most often was followed with supportive therapy without heparin.

Abnormal Venograms—

(1) **Filling defect**—The finding of a lucency within the lumen of an opacified vein is conclusive evidence of phlebitis. The term phlebitis is used because it is impossible to distinguish between thrombophlebitis and phlebothrombosis radiographically. The clot may or may not be adherent to the intima. A tail of clot often was seen as a tapered lucency extending proximally in a threatening manner.

Many of these patients had surprisingly mild pain and tenderness although definite "cords" were clinically palpable in some. Some patients had such serious diseases as carcinoma, while others had been immobilized for long periods. This change represents the acute stage of the natural course of the disease and usually lasts several days to one week. (Figure 1)

(2) **Venous occlusion**—It has been shown that clots become adherent to the vein wall and gradually occlude the lumen over the next one to two weeks. (Figure 2) The roentgen signs of occlusion are direct visualization of a cut-off of contrast medium within the lumen of a vessel. This can be seen at various levels in the leg. The popliteal vein at the knee joint, or the superficial femoral vein at the "adductor canal level" are common sites of occlusion. Deep leg veins and common femoral occlusion are less frequent. (Figure 3) Large multiple venous collaterals bypass the occluded segments of the deep system of veins. These can be differentiated from varicosities by their lack of extreme tortuosity and their location within muscle groups. Variocosities are more superficial and fill via perforating branches. These collaterals arise from a localized area where the lumen is patent, and are directed by the shortest available routes to the next segment of patent deep venous structure.

(3 **Non-opacification of deep venous structures**—Technical errors can be avoided with tight application of one or two tourniquets to eliminate filling of the superficial veins with contrast medium. Attention at the venipuncture site during injection will avoid extravasation due to back pressure with risk of skin slough. Segments of the deep venous structures may partially opacify to give a "ghost" appearance. A repeat

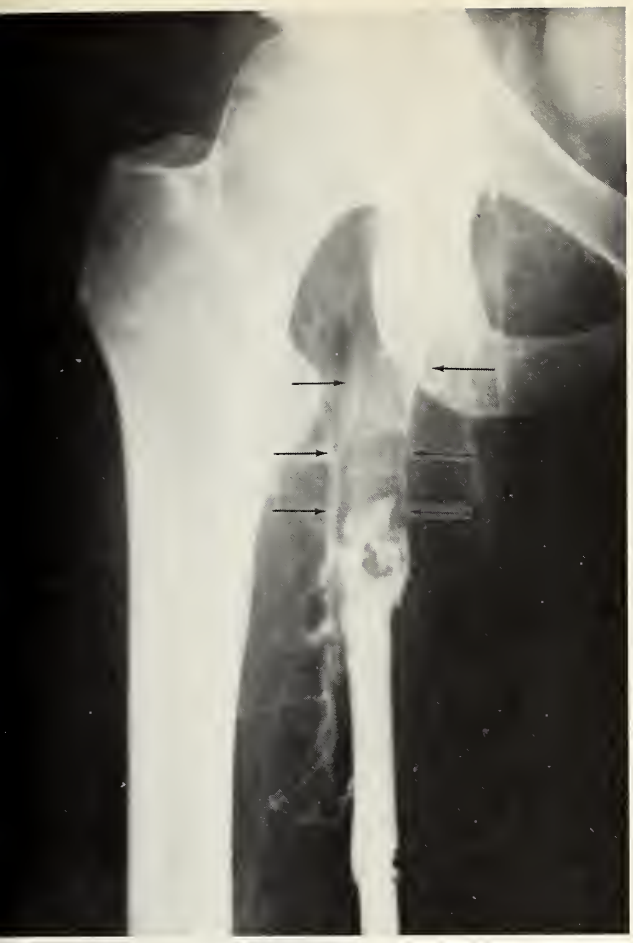


Figure 3—Venous occlusion of the common femoral vein corresponding to a two-week history of clinical pain and swelling of the extremity.



Figure 4—Non-opacification of all deep venous structures with return flow via collaterals. This patient was symptomatic for over three weeks.

injection after changing position and tightness of the tourniquets will corroborate nonfilling of veins. Large collaterals are seen especially in the muscular branches of the gastrocnemius and soleus muscles. These are the cases in which lateral filming is most useful in order to prove location of the muscular branches and demonstrate nonvisualization of deep veins. Cases of lower extremities completely drained by collateral veins require a presumptive diagnosis of total occlusion of deep venous structures which must be made on indirect evidence. The age of the inflammatory process can be estimated at three to four weeks. (Figure 4)

(4) **Recanalization**—The natural history of thrombophlebitis is recanalization of the venous lumen. This is represented radiographically by irregular linear densities indicating adhesive thrombus or synechiae outlined by contrast up against the vein wall. The valves may be distorted or destroyed and the path of contrast flow is interrupted by right-angle turns. Multiple channels may replace a single previous structure. Few collaterals are present at this time in the process, usually over one month since the onset of symptoms.

DISCUSSION

The increasing yield of abnormal venograms in the literature should encourage more widespread use of a very accurate procedure.[2,3,5] Newer methods of diagnosis are always being compared to venography and found less accurate. Many centers perform complicated maneuvers requiring patients to stand semi-erect, non-weight bearing while being subjected to infusion of large amounts of contrast and time-consuming fluoroscopy and spot filming.[8,13] We have found that the simplest and fastest procedure will demonstrate significant pathology adequately when considering scheduling and effort limitations of a busy angiography section.[6,7] Since fluoroscopy is not needed, this technique reduces radiation exposure and is particularly useful in patients requiring repeated serial studies. Many patients cannot tolerate the more complicated maneuvers employed because of weakness, dizziness, pain and swelling of the extremity, paralysis, decubitus ulcers, or recent surgical incisions.

The limitations of our procedure relate to missing small clots in superficial veins or small muscular veins. Superficial phlebitis is a diagnosis easily made on clinical signs; it is not life-threatening and does not require anticoagulation. The goal of emergency deep venography is quickly to identify those patients harboring sources of life-threatening pulmonary emboli in their legs. Iliac venography and inferior vena cavagraphy best are performed via femoral vein puncture since the iliac veins are opacified with this technique in only twenty-five percent of cases. The most elusive source of significant thrombi is a large network of deep pelvic and presacral veins. Investigation of pelvic varices is impossible, except by retrograde catheterization methods. There is no practical information derived from visualizing every tiny vein in the lower extremities when huge clots in pelvic veins remain an undetectable reservoir of potentially fatal pulmonary emboli.

The venogram quickly can provide vital accurate information about the natural progression of phlebitis. Filling defects, occlusions, nonopacification, and recanalization of veins as seen at venography reflect the time sequence of events that occur with phlebitis. This enables the clinician to choose a regimen of treatment confidently and avoid dangerous complications. [4,10,12]

SUMMARY

The "most advocated technique of venography" is to use no tourniquet, inject 100 to 200 cc. of contrast under fluoroscopic control with the patient balancing on one foot in the semierect position. This technique is difficult to perform on many patients.

A simpler, fast technique is advocated which leaves the patient supine, and utilizes the injection of 50 cc. of contrast with a tourniquet applied to the leg, and employs only overhead films. This deliberately ignores the superficial veins which are of no interest in the prevention of pulmonary emboli, but focuses attention on the deep system of veins. The technique encourages immediate requisition of venography for any patient with leg signs and symptoms suggestive of phlebitis.

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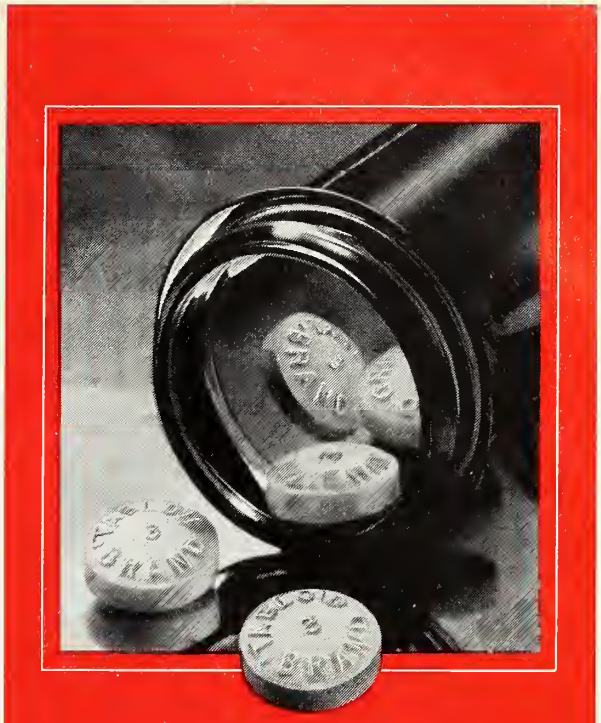
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Adult respiratory distress syndrome secondary to miliary tuberculosis activated after a complicated pancreaticodocholithotomy in a previously gastrectomized patient is presented. Activation of pulmonary tuberculosis in a malnourished patient following abdominal surgery, especially subtotal gastrectomy, may be anticipated. In contemplation of resectional or bypass gastrointestinal surgery, prophylactic use of isoniazid should be considered with inactive pulmonary tuberculosis. Long-term, postoperative follow-up, with special attention to the development of weight loss or malnutrition, also is advisable. Although miliary tuberculosis rarely is associated with adult respiratory distress syndrome, its early recognition is mandatory in order to initiate specific and effective therapy.

Adult respiratory distress syndrome secondary to activated miliary tuberculosis following recent pancreatic duct surgery and remote subtotal gastrectomy encompasses a rare combination of events and circumstances.

The reactivation of pulmonary tuberculosis following subtotal gastrectomy first was suggested by Winkelbauer and Frisch[1] in 1927 and has been reported subsequently in the European and American literature.[2-8] Retrospectively, an incidence up to 6.3 percent of pulmonary tuberculosis following subtotal gastrectomy has been reported.[7] The statistical significance is confusing because analyses vary as to whether patients were operated on with active tuberculosis, or with reactivation of old static lesions, or with development of tuberculosis in a healthy lung. In 1977, Yu reviewed seven cases of tuberculosis in which the onset followed intestinal bypass surgery for obesity.[9] Only three of these patients developed pulmonary tuberculosis; the other four had tuberculosis at extrapulmonary sites. It has been postulated that malnutrition is a common denominator for all cases of tuberculosis following surgery.[5]

Tuberculosis, as a cause for acute respiratory distress syndrome, is a rarity. To date, only twelve cases of adult respiratory distress syndrome secondary to pulmonary tuberculosis have been documented in the literature.[10-15] A mortality of 58 percent mandates a need for earlier diagnosis and more intensive treatment.

CASE REPORT

A 56-year-old male was transferred on 12/27/77 to the St. Barnabas Medical Center Intensive Care Unit for treatment of acute adult respiratory distress.

In 1959, the patient had a Billroth II subtotal gastrectomy for acute and chronic duodenal ulcers. Since then he was hospitalized on eleven occasions for acute and chronic relapsing pancreatitis including pseudocyst formation, drainage of a pancreatic abscess, cholecystectomy, repair of a wound dehiscence, and appendectomy. During that period, chest roentgenograms revealed a stable pattern of localized fibrocalcific densities in the apex of the left upper lobe and left hilum. The patient also had a hiatus hernia and chronic peptic esophagitis.

On 11/25/77, at Monclair Community Hospital, a laparotomy was performed for chronic recurrent pancreatitis. An eight mm calcium phosphate stone was removed from a pancreatic duct through the duodenal stump. Postoperatively, the patient developed a right pleural and subphrenic effusion containing 3+ bile and 250 units of amylase per 100 ml. Repeated serum amylase studies were normal. "Acute pulmonary congestion" responded temporarily to

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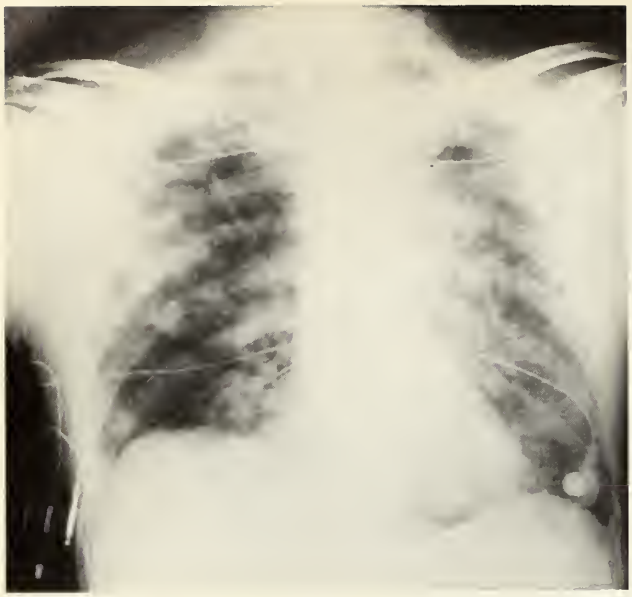


Figure 1—Chest roentgenogram illustrates the presence of bilateral alveolar infiltrates.

digoxin and furosemide. On 12/15/77, the chest roentgenogram disclosed bilateral diffusely disseminated alveolar infiltrates (Figure 1). The patient developed acute respiratory distress with sepsis and toxicity (temperature 105°F). Respirations had to be assisted and controlled on a volume cycled respirator. The pulmonary condition deteriorated despite antibiotics (chloramphenicol, ampicillin, gentamycin, amoxicillin) and nutritive care (3.5 percent amino acids, 10 percent intravenous fat emulsion, 25 percent glucose in 5 percent amino acid hyperalimentation solution). Cultures of blood, sputum, and pleural, subphrenic, and spinal fluid were negative except for E. coli in one sputum specimen. Multiple urine specimens contained Candida. The patient was transferred to St. Barnabas Medical Center Intensive Care Unit in a moribund condition.

Initial assessment of hemodynamic functions and oxygen transport by the Automated Physiologic Profile[16] revealed an elevated aortic pressure (205/87 with a mean of 124), a low cardiac output, tachycardia, diminished stroke index and left ventricular stroke work, increased total peripheral resistance, elevated mean aortic pressure, and a pulmonary artery wedge pressure of zero. This was compatible with hypertension in a hypovolemic state.

After fluid volume replacement over a 24-hour period, the physiologic profile (Figure 2) revealed a cardiac index 6 L/min/M^2; pulse 127/min; stroke index 48 ml/M^2; left ventricular stroke work 52 gm-M/M^2; total peripheral resistance 1000 dyne-sec-cm^{-5}-M^{-2}; right ventricular stroke work 5 gm-M/M^2; oxygen consumption 230 ml/min/M^2; veno-arterial admixture Q_s/Q_t 26%; serum lactate 12 mg/dl; arterial base excess +4 meq/L; pulmonary artery wedge pressure 0 mmHg; pulmonary artery pressure 23/0 with a mean of 7 mmHg; aortic pressure 136/66 with a mean of 80 mmHg. The elevated cardiac index, tachycardia, diminished peripheral resistance, wide pulse pressure, and moderate decrease in mean aortic pressure were compatible with a hyperdynamic cardiovascular response associated with the early phase of septicemia. The elevation in cardiac output resulted in a high oxygen consumption which is also characteristic of the early phase of sepsis. The elevated intrapulmonary shunt correlated with the infiltration in the

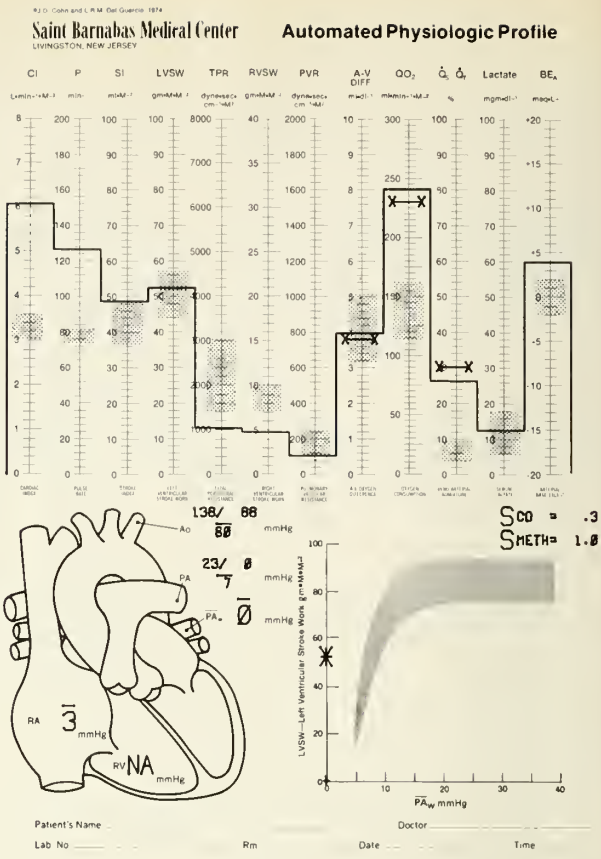


Figure 2—The automated physiologic profile performed after volume expansion demonstrates the presence of a hyperdynamic circulatory state. The hemodynamic and oxygen transport data are consistent with septicemia.

portable chest roentgenogram and the acute respiratory distress.

Respiratory control was maintained on the MA-1 respirator with FIO_2 of 0.50. Serum lactic acid and arterial base remained normal, indicating satisfactory peripheral perfusion. Infusion of 8,780 ml in 48 hours raised the pulmonary artery wedge pressure to four mmHg. Repeat studies were compatible with a hyperdynamic state and sepsis.

Evaluation for a focus of infection with ultrasound, liver and lung scans, body gallium scan, echocardiogram, gastrointestinal series and repeat laparotomy were not diagnostic. The sepsis and toxicity were refractory to massive supportive care including chloramphenicol, clindamycin, ampicillin and gentamycin. On 12/31/77, through a right thoracotomy, biopsies were taken of 0.5 to one cm nodules disseminated throughout the right lung. Pathology studies demonstrated numerous acid-fast micro-organisms in necrotizing bronchopneumonia. Isoniazid and ethambutol were added to gentamycin for therapy of pulmonary tuberculosis. When the patient lapsed into a coma and was suspected of having tuberculous meningitis, supplementary dexamethasone was given.

Coagulation profile on 1/1/78 was normal except for elevated fibrin split products. On 1/3/78, an immunocompetence profile (figure 3) demonstrated a modest systemic response manifested by elevation in the erythrocyte sedimentation rate to 34 mm/hr. Immunoglobin IgG, IgA, IgM and complement C3 and C4 fractions were within normal limits. Cell mediated immunity studies revealed several abnormalities. The total lymphocyte count was diminished to 8000 mm^{-3} and was associated with a decrease in the B-cell

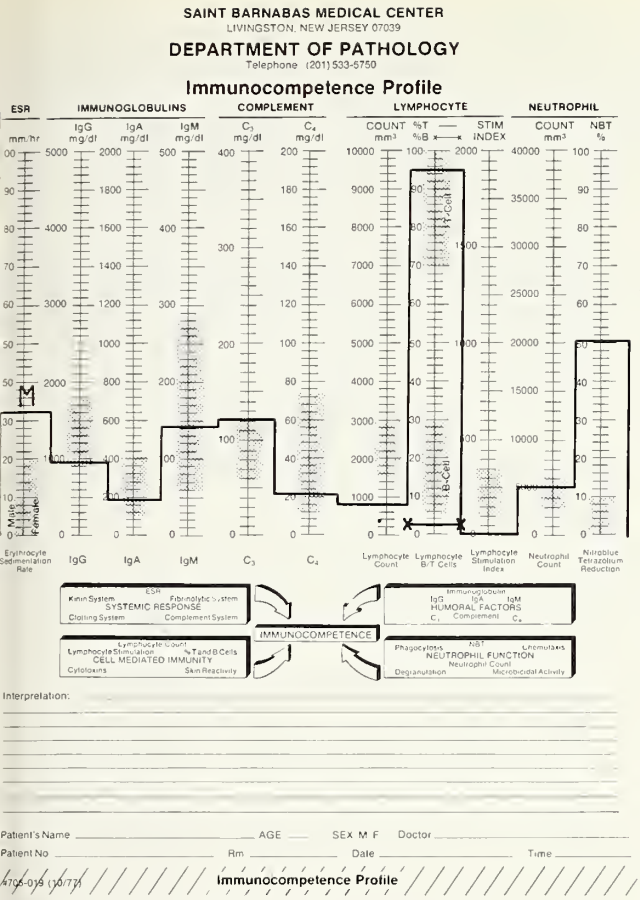


Figure 3—Immunocompetence evaluations demonstrate the presence of normal values for humoral factors. Depressed lymphocyte stimulation is related to steroid administration and depressed nutritional function.

traction. The absence of lymphocyte stimulation reflected malnutrition and steroid therapy.[17] Neutrophil function as assessed by the neutrophil count and the nitroblue tetrazolium test demonstrated an endotoxin response.[18] This is not in conformity with gastrectomized tuberculosis patients who do not increase their leukocytes during extended physical stress compared to non-gastrectomized patients.[19]

Additional complications which the patient survived were bloody enterocolitis, occlusive thrombophlebitis of the subclavian vein with massive swelling of the left upper extremity, and cystitis. Because of a lack of infusable veins, nutrition was sustained by gastric tube feedings. A nutritional profile on 2/1/78 revealed major deficiencies in visceral protein and cellular immunity, consistent with the chronic catabolic clinical course.[20] After an antituberculosis regimen consisting of INH, ethambutal, rifampin and pyrazinamide the smear and culture of the sputa became negative. The patient resumed normal activities in August, 1978.

DISCUSSION

It long has been accepted that the reactivaion of tuberculosis occurs in a setting with a depressed immune system. Gastrectomized patients, particularly those with a dumping syndrome, often suffer from malnutrition secondary to intestinal malabsorption. Reduction of intestinal absorption and serum proteins result in an increase in tubercle bacilli in the sputum and more frequent relapses of pulmonary tuberculosis.[5]

Pulmonary tuberculosis usually is not associated with acute respiratory failure. Recognition that miliary tuberculosis may cause an acute respiratory distress syndrome is important since specific and effective therapy for the disorder is available. However, an early diagnosis may be difficult to secure unless a high index of suspicion is maintained. This especially pertains to acute and chronic debilitating states. In the present case with a subtotal gastrectomy and chronic relapsing pancreatitis, pancreaticodocholithotomy with a postoperative duodenal stump leak was an antecedent factor. The acute respiratory distress syndrome was refractory to broad spectrum antibiotic therapy until a pathologic diagnosis of miliary tuberculosis was made from an open lung biopsy. Then, sputa were examined for acid-fast bacilli and were found to be positive. A tissue diagnosis may have to be resorted to in miliary tuberculosis since only 20 to 40 percent of patients have positive sputa smears. Also, the second strength PPD test was negative. A false negative skin test is found in 30 to 60 percent of miliary tuberculosis cases.[14].

Evidence of a consumptive coagulopathy was present in seven of the twelve documented cases in the literature. Six of these seven cases died. Except for the slightly elevated fibrin split products, there was no coagulopathy in the present case.

Postoperatively, when the patient lapsed into a coma secondary to suspected tuberculous meningitis, a short course of dexamethasone was used to advantage in conjuction with the antituberculosis regimen. Although controversial in the management of tuberculosis, corticosteroid therapy appears to be effective in reducing systemic toxicity and the intensity of the inflammatory exudative pulmonary response.[14] The literature dealing with the use of corticosteroids in adult respiratory distress syndrome is mostly anecdotal and no firm recommendations can be given. [21]

CONCLUSION

1. Adult respiratory distress syndrome secondary to miliary tuberculosis activated after a complicated pancreaticodocholithotomy in a previously gastrectomized patient is presented.

2. Guidelines as provided by the automated physiologic profile, immunocompetence profile, and nutritional profile in the management of this patient are discussed.

3. Activation of pulmonary tuberculosis in a malnourished patient following abdominal surgery, especially subtotal gastrectomy, may be anticipated. In contemplation of resectional or bypass gastrointestinal surgery, prophylactic use of isoniazid should be considered with inactive pulmonary tuberculosis. Long-term, postoperative follow-up is also advisable with spécial attention to the development of weight loss or malnutrition. Although miliary tuberculosis rarely is associated with adult respiratory distress syndrome, early recognition is mandatory in order to initiate specific and effective therapy.

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Rhinoscleroma Presenting as Upper Airway Obstruction

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A case of rhinoscleroma presenting with repeated episodes of dyspnea due to upper airways obstruction is described.

Rhinoscleroma is a chronic, progressive, granulomatous disorder involving the mucosa of the respiratory tract and caused by the bacteria Klebsiella rhinoscleromatis. Granulomatous involvement of the larnyx and trachea may produce upper airway obstruction. The purpose of the report is to describe a patient who presented clinically with recurrent dyspnea and who was found to have rhinoscleroma involving the larynx.

CASE REPORT

A 20-year-old female was referred to the Saint Michael's Medical Center Asthma Clinic with a chief complaint of wheezing and dyspnea. The patient had been treated for asthma with intermittent bronchodilators for the past ten years. Three years ago she was hospitalized for dyspnea associated with stridor and required endotracheal intubation and controlled ventilation. A diagnosis of status asthmaticus and acute laryngeal spasm was made. One year prior to admission she noted increasing dyspnea on exertion, nonproductive cough, and wheezing with decreasing responsiveness to bronchodilators. Two weeks prior to admission she became dyspneic at rest.

The patient was born in Guatemala and emigrated to the United States ten years prior to admission, but made frequent return trips to her native country.

Physical examination revealed a well-developed, well-nourished female in moderate respiratory distress with stridor. Her respiratory rate was 32 per minute and pulse 92 beats per minute. Examination of the nose, mouth, and throat was unremarkable. Auscultation of the chest revealed bilateral diffuse inspiratory and expiratory wheezes heard maximally over the sternal notch. The heart and abdominal examinations were normal.

Radiographs of the chest were normal. Forced expiratory volume (FEV) was 1.0 liters and forced vital capacity (FVC) was 1.5 liters, with no reversibility following bronchodilators. Flow volume loop demonstrated both a flat inspiratory and expiratory loop characteristic of fixed airway obstruction. Laminograms of the trachea demonstrated a 90 per cent occlusion of the trachea by a 13 millimeter polypoid lesion in the subglottic space one millimeter below the vocal cords. (Figure 1) Bronchoscopy and excision of the mass were performed. The histopathology was diagnostic of rhinoscleroma (Figures 2, 3), and culture of the lesion revealed Klebsiella ozaenae. A flow volume loop one week following surgery was normal with no evidence of extrathoracic obstruction. A course of streptomycin and tetracycline was started and the patient became asymptomatic.

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Figure 1—Laminograms of the trachea demonstrating a 13 mm polypoid lesion subglotically.

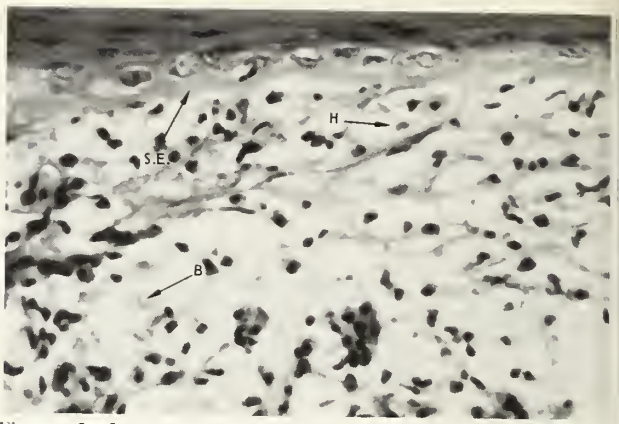


Figure 2—Laryngeal tissue lined by metaplastic squamous epithelium, beneath which are large histiocytes (Mikulicz cells) with small nuclei and abundant foamy cytoplasm containing bacilli. There is a focal infiltrate of plasma cells and lymphocytes. Hematoxylin and eosin. x400

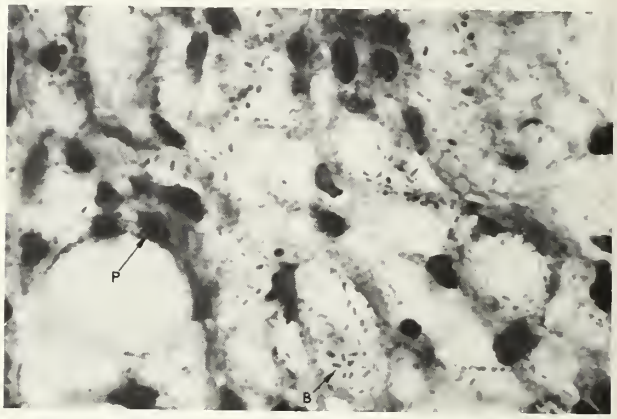


Figure 3—Histiocytes with cytoplasm containing numerous gram negative bacilli. Several plasma cells are present. Taylor's stain x 1,000.

DISCUSSION

Rhinoscleroma is endemic to Central America, southwest Russia, Czechoslavakia, Poland, the Balkan countries, Italy, North and East Africa, India, and Indonesia.[1] Our patient was from Guatemala, an area with probably the highest incidence of rhinoscleroma in the world. Rhinoscleroma is rarely seen in the United States but with jet age travel and migration, its incidence may increase.

Initial symptoms are similar to a common cold, with malaise, fever, and leukocytosis. It is because of the formation of nodular proliferative granulomas that most individuals seek medical attention. After several years these scleromata undergo fibrosis with the development of extensive scar tissue, visible contraction, and deformities.[2] Scleroma may be found anywhere in the respiratory tree from the nose to the major bronchi. In a series of 241 cases reported by Semczuk, one hundred per cent had involvement of the nose.[3,4] Our case is distinctly unusual in that there was no nasal involvement. Acuna, in a large series, reported involvement of the larnyx in fifteen percent of cases.[5] The trachea and bronchi were involved in two percent.

Scleroma of the larynx and trachea is secondary to lesions in the upper respiratory tract. Primary scleroma of the larynx is an exceedingly rare lesion.[6] Scleroma involving the larynx may produce changes in voice, including hoarseness, and cough productive of small amounts of thick red sputum. The most distressing and dangerous symptom is dyspnea which occurs when there is significant upper airway obstruction. Our patient had symptoms misconstrued as asthma for many months prior to her admission.

The diagnosis of rhinoscleroma is confirmed by biopsy demonstrating the characteristic histopathology and by culturing Klebsiella rhinoscleromatis. Ssali, by culturing biopsy material on blood agar, found Klebsiella rhinoscleromatis in ninety-eight percent of all cases.[7] Microscopically the Mikulicz cell, a foamy vacuolated histiocyte in which gram negative diplobaccilli can be seen in the cytoplasm, is fairly characteristic.

Treatment is both medical and surgical. When scleroma involves the lower respiratory tract, treatment with streptomycin and tetracycline is recommended; periodic retreatment may be necessary. Of eleven patients seen at the University of Illinois Hospital with lower respiratory disease, nine required tracheostomy some time during the course of their illness.[8] In our case a single polypoid lesion was found which was surgically excised.

Finally, any individual who is from or has visited an endemic area and presents with upper airway obstruction with nasal pathology should be considered a possible victim of rhinoscleroma.

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Mediastinal Tumor Presenting as a New Heart Murmur: Diagnosis and Treatment*

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Because of their strategic location mediastinal tumors are potentially lethal and can produce a wide variety of signs and symptoms. The following report illustrates how successful diagnosis and treatment of a benign teratoma in a 16-year-old female depended on a rational combination of clinical evaluation, chemotherapy, repeat C-T scanning during therapy, and surgery.

The finding of a heart murmur not heard previously is always alarming and demands a prompt explanation. Infective endocarditis, torn chordae tendinae, traumatic ventricular septal defect, ruptured papillary muscle following acute myocardial infarction, and acute ventricular enlargement are some of the more common causes of a new heart murmur. All of these require immediate medical or surgical treatment. An uncommon cause of a loud heart murmur that previously was absent is the impingement on the aorta and pulmonary outflow tract by an expanding mediastinal tumor. The following is a description of the successful diagnosis and treatment of a mediastinal teratoma in a formerly healthy 16-year-old girl.

CASE REPORT

A 16-year-old female was well until October 1977, at which time she experienced intermittent lumbar and thoracic midline back pain. The pain was unrelieved by salicylates and became progressively severe and prolonged. Subsequently the patient developed severe nonradiating, anterior chest pain, dyspnea at rest, malaise, and anorexia, with a ten-pound weight loss over the next month. She was seen by her physician who noted a temperature of 101 degrees F. and a Grade III/VI systolic murmur that had not been present before the onset of symptoms. The patient was admitted to St. Michael's Medical Center for evaluation of possible infective endocarditis.

The patient and her parents specifically denied any previous cardiac symptoms, past rheumatic fever, dental manipulation, or intravenous medication. She had been seen for minor lower respiratory tract infections by her physician who in the past had never heard any abnormal cardiac sounds. She had participated in gymnasium activities at school until one month prior to admission.

On physical examination the patient presented as a well-developed, well-nourished female complaining of diffuse pleuritic anterior chest pain. The pulse was 100 and regular, blood pressure 130/70 without pulsus paradoxicus, respiratory rate was 16, and the temperature was 100.8 degrees F. Examination of the head, eyes, ears, nose, and throat was normal. The neck was supple with normal jugular venous pulse and bilateral transmitted carotid bruits. One right anterior cervical lymph node was identified. It measured one by one centimeter and was non-tender, soft, and movable. The chest wall was non-tender. The cardiac apex impulse was at the midclavicular line in the fifth intercostal space. S1 was normal. A Grade III/VI long systolic ejection murmur was heard at the second right and left intercostal space next to the

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Figure 1—PA chest x-ray demonstrates anterior mediastinal mass and normal cardiac size.

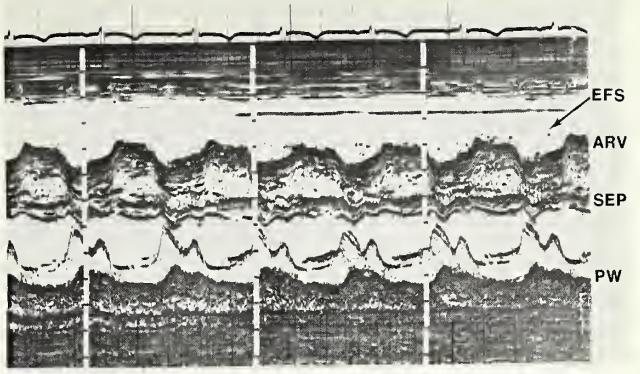


Figure 2—Echocardiogram demonstrates an anterior echo-free space suggestive of a tumor in the anterior mediastinum. ARV = anterior right ventricular wall; EFS = echo free space; SEP = interventricular septum; PW = left ventricular posterior wall.

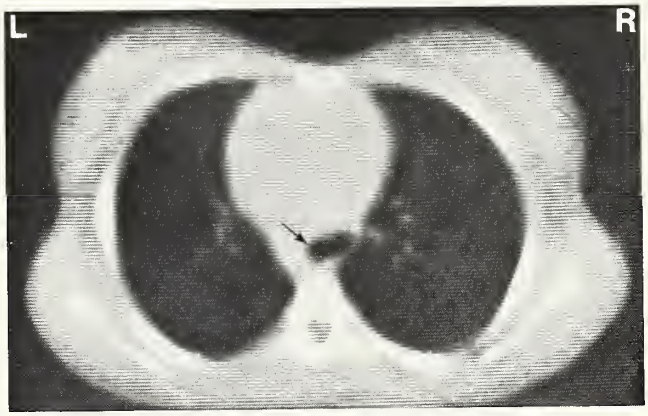


Figure 3—CT scan demonstrating occlusion of the left mainstem bronchus by extrinsic tumor compression. (Arrow indicates left mainstem bronchus)

sternum; it radiated widely to the neck and left shoulder. S2 was normal. No gallop sounds or diastolic murmur were heard. The lung fields were clear to percussion and auscultation. The remainder of the physical examination was entirely normal. The spleen was not palpable, and except for the lymph node noted above no adenopathy was found.

The hemoglobin was 10.0 grams, hematocrit 31.2% and white blood count 15,000 with 80% polys, 14 lymphs, 4 bands, and 2 monos. The erythrocyte sedimentation rate was 115 mm/hr. SMA-12 and protein electrophoresis were entirely normal. The electrocardiogram showed a sinus tachycardia, rate 100 per minute, axis +75 degrees and minor non-specific ST segment and T Wave abnormalities. Chest x-ray (Figure 1) demonstrated a large anterior superior mediastinal mass of apparent homogeneous density. The echocardiogram (Figure 2) demonstrated an anterior echo free space suggestive of a mass lesion. The left and right ventricular dimensions, the left atrial dimension and aortic root dimension were all normal.

A low-grade fever persisted and multiple aerobic and anaerobic blood cultures were negative thus eliminating the diagnosis of infective endocarditis. The chest pain was partially relieved with salicylates.

Because the mediastinal mass obscured the radiographic cardiac contour, a gated blood pool scan was performed and demonstrated the mediastinal mass and also suggested a pericardial effusion. The cardiac outline was normal. Gallium scan confirmed the presence of the mediastinal mass with gallium uptake and demonstrated no additional areas of uptake or radioactivity.

Although the clinical presentation strongly suggested a malignant lymphoma, histologic confirmation was mandatory before starting treatment. The cervical lymph node found on admission was considered for biopsy, however on reexamination it was felt not to be pathologic. Therefore on the tenth hospital day an exploratory mediastinoscopy was carried out under general anesthesia in order to obtain tissue for microscopic examination. During the procedure the left lung could not be aerated, despite partial withdrawal of the endotracheal tube. Postoperatively, in the recovery room, the patient was cyanotic and remained unconscious. Arterial blood gases on an FIO_2 of 100% revealed pH 6.87, PO_2 152 mm, PCO_2 101 mm, and bicarbonate 17.2 meq/L. Diagnosis of severer espiratory acidosis secondary to left mainstem bronchus obstruction was suspected and emergency fiberoptic bronchoscopy confirmed that the left mainstem bronchus was completely occluded by extrinsic compression, presumably by the tumor mass. Intravenous glucocorticosteroids and repositioning the patient into a sitting position led to rapid normalization of blood gases and the patient awoke without any neurologic sequelae.

Histologic review of the lymph node removed at mediastinoscopy revealed no abnormal histology. A thoracic CT scan was performed and demonstrated a homogeneously dense mediastinal mass that nearly completely occluded the left mainstem bronchus (Figure 3). In order to shrink the tumor mass enough safely to permit reexploration by open thoracotomy, yet not so much that the cellular architecture of the mass would be distorted to a degree that would preclude histologic diagnosis, the patient was treated with hydrocortisone, two grams intravenously daily in divided doses, followed by prednisone 40 mg orally daily for 15 days and mediastinal radiation with the total dose of 700 rads in divided doses. Repeat CT scan (Figure 4) performed after therapy revealed that the left mainstem bronchus was now widely patent and that the tumor mass was significantly smaller. The patient then underwent open exploratory right thoracotomy without any intra or postoperative complications. At surgery a large cystic teratoma partially adherent to the pericardium was found. The majority of the tumor was resected, and all remaining tissue that could not be removed was macerated to prevent further growth. Histologic review



Figure 4—CT scan following mediastinal radiation and steroid therapy. Note that both mainstem bronchi are now widely patent. Compare with Figure 3. (Arrow indicates left mainstem bronchus)



Figure 5—Postoperative chest x-ray. No evidence of mediastinal tumor is seen. Compare with Figure 1.

of the excised tissue confirmed the diagnosis of a benign teratoma, with evidence of glandular tissue, hair follicles, epidermis, and muscle cells. Postoperative chest x-ray is shown in Figure 5.

Postoperatively the patient recovered uneventfully and six months after discharge is completely asymptomatic. The murmur present before open thoracotomy disappeared after removal of the tumor and has not recurred.

DISCUSSION

This case demonstrates several unusual features. First, extrinsic compression by a mediastinal mass on the great vessels as a cause of a pulmonic outflow murmur is quite uncommon; fewer than 30 cases have been described.[1,2] Winter *et al.* postulated that the ease with which an anterior mediastinal tumor can expand laterally accounts for the uncommon finding of great vessel distortion and consequent generation of a heart murmur.[3] No physical finding is specific for this abnormality, however Littler noted disappearance of the murmur due to tumor compression with the inspiratory phase of respiration in three of his four cases.[4] This is in contradistinction to the inspiratory increase of the systolic murmur of valvular pulmonic stenosis with which it could be confused. Inspiratory disappearance of the murmur caused by extrinsic pressure on the pulmonary artery might be accounted for by the known increasing volume of the thorax with inspiration which would lift the tumor off of the great vessels [4]. In our case espiratory variation was absent, presumably because the tumor was sufficiently large to compress the great vessels throughout the respiratory cycle.

The mechanism for the acute bronchial obstruction during mediastinoscopy is unclear, however mechanical irritation by the endotracheal tube used for anesthesia and/or the anesthetic agent itself in conjunction with compression of the mainstem bronchus by the tumor probably were contributing factors. Position of the patient and the fact that she was young with soft bronchial rings may have played a role. In retrospect, the lateral chest film demonstrated encroachment of the tumor on the left mainstem bronchus.

Computerized tomography played a unique role in the management of this patient. The degree of bronchial constriction by the tumor was documented by the initial CT scan and, after radiotherapy and treatment with corticosteroids, relief of the bronchial compression by the tumor was demonstrated by a repeat comparative CT scan. After expansion of the left mainstem bronchus was demonstrated, thoracotomy could be carried out safely. Of interest is the CT image of the tumor itself; the tumor appeared homogeneous in density suggesting a diagnosis other than a teratoma which is characteristically heterogenous on gross and microscopic examination. In at least one other reported case a similar discrepancy was found; a heterogenous mediastinal teratoma appeared to be homogeneous on the CT scan, thus suggesting a diagnosis of lymphoma[5].

The other unusual feature of this case is the severity and abundance of symptoms caused by the tumor. Not only were local complications encountered (bronchial obstruction, chest pain, dyspnea, and a heart murmur) but the patient also had systemic symptoms of fever to 101, malaise, back pain, and a ten-pound weight loss. These systemic manifestations in conjunction with the new heart murmur, led to the initial suspicion of endocarditis followed by the suspicion of malignancy. Although not commonly associated with a benign teratoma, similar systemic signs and symptoms are not uncommon in patients with any type of mediastinal space-occupying lesion. Even though it was felt strongly that the patient's tumor was most likely malignant, the diagnosis could only be made definitely by histologic review of the tissue. This case demonstrates the absolute necessity of making a histologic diagnosis in cases of mediastinal tumors before radiation in tumoricidal doses or chemotherapy is undertaken.

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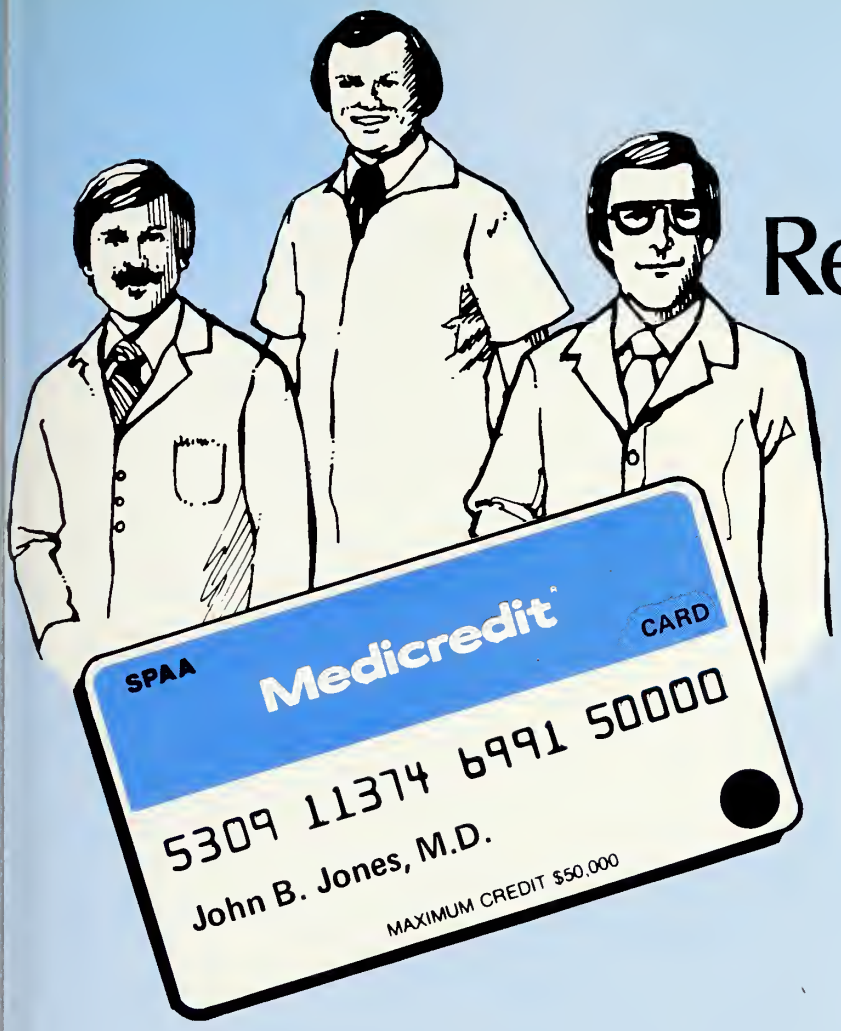
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Incidental Finding of ^{67}Gallium Accumulation in a Ventral Hernia.*

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An unusual appearance of ^{67}Gallium citrate within a ventral hernia is presented. The need of careful clinical correlation and ancillary studies with such scans is strongly emphasized.

In this report we will describe the incidental finding of ^{67}Gallium citrate accumulation in a large incarcerated ventral hernia. The bizarre appearance of the ^{67}Gallium citrate activity on the anterior photoscan with a normal appearing posterior photoscan might be misdiagnosed without correlation with a barium enema. This case reveals once again the inherent problems in interpretation of ^{67}Gallium photoscans of the abdomen without careful clinical and radiological correlation. To our knowledge this unusual appearance has not been described previously.

CASE REPORT

A 59-year-old obese female was admitted for oncological evaluation and treatment of a previously diagnosed stage I adenocarcinoma of the uterus. Her past and family histories were non-contributory except for a long-standing history of hypertension. The physical examination was negative except for the patient's large abdominal girth.

The oncological survey consisted of an excretory urogram, bone scan, and ^{67}Gallium citrate tumor detection scan. The studies were within normal limits except for a bizarre-appearing ^{67}Gallium frontal photoscan (Figure 1) with a normal posterior photoscan (Figure 2). A follow-up barium enema revealed a large ventral hernia incorporating almost the entire transverse colon (Figure 3).

DISCUSSION

Since the original report of ^{67}Gallium citrate use for tumor detection by Edwards and Hayes in 1967, the localization of this radionuclide has been shown to include a variety of non-neoplastic lesions, such as abscesses[1], pulmonary sarcoidosis[2], myocardial infarctions[3], empyema of the gallbladder[4], inflammatory and neoplastic pulmonary processes[5], and even in a thrombosed vein[6].

Many authors have claimed that the most vexing problem of ^{67}Gallium photoscanning is the presence of large-bowel activity obscuring abnormal pathology[7,8]. Enemas plus repeat scanning frequently must be resorted to for evaluation of the abdominal photoscans. Normal ^{67}Gallium activity at 48 hours is concentrated in the axial skeleton, liver, spleen, and large joints. Activity often can be recognized in the salivary, lacrimal, and mammary glands[7]. Nelson *et al.*, using autopsy specimens, found fecal material which had greater activity than tumor or other tissue.[9]

This problem has lead Paterson and McGrady to suggest the use of alternate agents such as ^{111}Indium-labeled bleomycin to detect intra-abdominal lesions because of its renal rather than intestinal pathway of excretion[10].

In our case the unusual appearance of ^{67}Gallium activity on the anterior scan only is not clear until one correlates it with the barium enema. It is noted that the ^{67}Gallium in the large bowel incorporated in the hernia takes a configuration similar to that seen in the barium enema study.

Careful clinical and radiological correlation of ^{67}Gallium

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Figure 1—Anterior ^{67}Gallium photoscan revealing a bizarre pattern of activity reaching down into the pelvis.



Figure 2—Normal posterior ^{67}Gallium photoscan. The large solid arrow indicates the liver, the small solid arrow the spleen, and the open arrow the axial skeleton.



Figure 3—A barium-enema film revealing the transverse colon incorporated in a large ventral hernia.

photoscan interpretation, especially with regard to abnormal abdominal foci of activity, remains a necessity of good radionuclide technique.

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REVIEW ARTICLE

A Review of Countercurrent Immunoelectrophoresis

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Countercurrent immunoelectrophoresis (CIE) rapidly is developing into a useful laboratory procedure in the diagnosis of certain bacterial, viral, fungal, and parasitic infections. The diagnostic applications of CIE for infectious diseases are reviewed. Some problems associated with the interpretation of the results also are mentioned.

The diagnosis of bacterial infection remains a constant problem to the clinician. The search for a rapid, sensitive, specific, and technically feasible laboratory procedure for the diagnosis of bacterial infection continues. The standard bacteriologic methods have certain limitations since bacteria may be missed on examining gram-stained smears and organisms may not be recovered from cultures.[1] In addition, errors in the interpretation of gram-stained smears may be committed even by experienced observers.[2,3] Countercurrent immunoelectrophoresis (CIE) is a technique for the detection of capsular antigen using specific antisera, which evolved from the fusion of two separate developments in immunology.

DEVELOPMENT OF THE TECHNIQUE

In 1897 Kraus demonstrated a precipitation reaction between the clear filtrate of a bacterial culture and the serum of an animal previously injected with the same filtrate.[4] This immunoprecipitation technique became the basis for the detection of meningococcal antigen devised by Vincent and Bellot[5] using a polyvalent horse meningococcal antiserum against the supernatant of centrifuged spinal fluid from patients with meningococcal meningitis. The procedure required 6 to 12 hours to develop. In 1933, Rake[6] correlated the intensity of the reaction to the number of organisms in the spinal fluid and four years later Alexander[7] correlated the rapidity of the reaction to the prognosis of the disease.

In a separate development, Grabar and Williams demonstrated the electrophoretic separation of serum proteins in agar and the precipitation of the proteins with specific antisera by diffusion.[8] It became possible to identify distinct protein entities but the procedure resulted in blurred precipitation lines during the period of diffusion. Ressler improved the technique by incorporating homologous antiserum in the agar matrix producing narrower and more distinct curves of precipitation.[9] Five years later, Laurell modified this principle and established the prototype of the technique of countercurrent immunoelectrophoresis.[10]

DIAGNOSTIC APPLICATIONS OF CIE

It was not until 1971 when the Laurell test or CIE was applied by Edwards in the detection of bacterial antigens.[11] The initial work was done on sera of 23 patients with meningococcal disease. Five of these patients who died within 24 hours of admission were positive by CIE. Since then, numerous reports on the use of CIE have appeared in the literature.

Dorff, Coonrod, and Rytel adapted the same technique to the detection of pneumococcal antigen.[12] Sera from 17 patients with positive blood cultures for S. pneumoniae were

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tested with Statens Seruminstitut (Copenhagen) omniserum containing 82 pneumococcal serotypes. Sera from three patients demonstrated strong lines of immunoprecipitation. The usefulness of CIE in detecting meningococcal disease was confirmed by Greenwood, Whittle, and Dominic-Rajkovic, when they reported the results of a field study done in Nigeria on 68 cases of suspected meningococcal meningitis.[13] Forty-seven cerebrospinal fluid (CSF) samples were positive by CIE, but only 45 CSF samples were positive by culture and gram-stain. When meningococcal antigenemia was investigated, the correlation was noted to be poor. Of 77 serum samples from suspected cases of meningococcal meningitis, ten samples gave a positive CIE. The test was positive in three of 14 patients with positive blood cultures and four of 42 patients with negative blood cultures. Coonrod and Rytel studied the CSF of 30 patients with pneumococcal, meningococcal or H. influenzae type b meningitis and they were able to detect antigen in 93 percent of patients.[14] They were the first to demonstrate the detection of H. influenzae type b capsular antigen by CIE in 19 patients with H. influenzae type b meningitis. There were no false-positive CIE reactions in the CSF of 17 controls. Edwards *et al.* later confirmed the feasibility of detecting H. influenzae type b capsular antigen by CIE in two infants with H. influenzae type b meningitis.[15] Ingram *et al.* also reported 14 additional cases of H. influenzae type b meningitis where 11 had positive CIE.[16] H. influenzae type b capsular antigen was positive in four of ten partially treated patients with meningitis, including two patients who initially had positive cultures for H. influenzae type b. Antigen was still detected after 24 hours and seven days of therapy with ampicillin in these two patients.

Aside from CSF and serum, other body fluids have been tested with CIE. Urine appeared to be a convenient specimen to look for capsular antigens. In pneumococcal pneumonia, pneumococcal capsular antigen can be detected in serum and urine in 57 percent of cases.[17] Several methods of concentrating urinary capsular antigen have been described in order to increase the yield of positive results.[18,19] In a brief report, Verhoef and Jones detected pneumococcal antigen in 14 sputum samples from 21 patients with positive sputum cultures for S. pneumoniae.[20] They homogenized sputa in pancreatin or trypsin and diluted it ten-fold to avoid prozones and to increase the sensitivity. Lampe *et al.* studied pleural fluid from 87 patients and found good correlation between culture and CIE for pneumococcus.[21] They noted that CIE was positive when gram-stained smears were positive. A cross-reaction between E. coli antigen and H. influenzae antisera was observed. They observed that pneumococcal antigen persisted in the pleural fluid for an average of ten days.

CIE initially was used for the detection of the capsular antigens of meningococcus, pneumococcus, and H. influenzae type b. The data are summarized in Tables I through IV. However, recently the procedure has been used for a variety of other organisms. Edwards and Larson used CIE to detect group B streptococci.[22] Bartram *et al.* reported

Table I
Data Review on CIE
Streptococcus Pneumoniae

Investigator	No. of Cases	CIE Positive	Culture Positive	Specimen
Dorff (1971)	17	3	17	Serum
Coonrod (1972)	7	7	7	CSF
Fossieck (1973)	6	5	6	CSF
Coonrod (1973)	11	5	11	Serum
	11	7	11	Urine
Bortrom (1974)	10	8	10	Serum
Verhoef (1974)	21	14	21	Sputum
Coonrod (1976)	46	19	46	Serum
Lompe (1976)	19	19	19	Pleural fluid

Toble II
Dato Review on CIE Neisseria Meningitidis CSF

Investigator	No. of Coses	CIE Positive	Culture Positive
Greenwood (1971)	68	47	45
Coonrod (1972)	4	4	4
Edwards (1972)	19	16	17

Toble III
Data Review on CIE Neisseria Meningitidis Serum

Investigotor	No. of Cases	CIE Positive	Culture Positive	Culture Negotive
Edwords (1971)	23	5	23	
Greensood (1971)	17	3	14	
	46	4		42

Table IV
Data Review on CIE H Influenzae Type b CSF and Serum

Investigator	No. of Cases	CIE Positive	Culture Positive	Specimen
Coonrod (1972)	19	17	19	CSF
Edwards (1972)	2	2	2	CSF
Ingram (1972)	14	11	14	CSF & Serum
Shackelford (1974)	31	24	31	CSF
	22	6	0	Partially treated CSF

the use of CIE in the detection of pseudomonas infections. Pseudomonas antisera were raised from rabbits immunized with 13 Habs types of pseudomonas. Antigen was detected by CIE in sera from 11 of 12 patients with pseudomonas infections. However, *in vitro* cross-reactions were noted. Nagel *et al.* showed the feasibility of detecting teichoic acid precipitin in the sera of patients with staphylococcal endocarditis by CIE using sonicated extracts of staphylocci.[23] It was positive in 25 of 26 cases of staphylococcal endocarditis. However, significant false positive reactions were noted in healthy and non-infected drug users and in patients with streptococcal infections. CIE has been used in the diagnosis of human brucellosis.[24] A protein antigen derived from brucella melitensis reacted with 94 percent of patients with brucellosis. Menonna *et al.* detected precipitating antibodies in mycoplasma infection by CIE.[25] The test appeared to be specific and was postive in patients with elevated levels of complement-fixing antibodies. In lepromatous leprosy, antimycobacterial antibodies were detected by CIE.[26] A treponema pallidum-CIE also has been developed to detect specific treponemal antibodies.[27] The sensitivity of T. pallidum-CIE was estimated to be 82 percent. There was agreement between T. pallidum-CIE and FTA-ABS in 96 percent of cases. CIE also has been used in the diagnosis of viral infections. The Australia antigen initially was tested by CIE but recently the procedure has been replaced by the radioimmunoassay (RIA) because of greater sensitivity of the RIA technique.[28,29] In cytomegalovirus (CMV) infections, a good correlation between CIE and the complement-fixation antibody test was noted.[30,31] CIE was positive in 14 (95 percent) of 15 patients with high titers of complement-fixing antibodies. The sensitivity and specificity of CIE was 88.9 percent. Using a purified chlamydia trachomatis protein antigen, CIE was positive in 43 (96 percent) of 45 cases of confirmed lymphogranuloma venereum (LGV) infections.[32] A CIE procedure has been developed for the following fungal infections: cryptococcosis, coccidioidomycosis and aspergillosis.[33-35] In cryptococcal infections, CIE detected 1.25μ gm/ml of cryptococcal antigen in the CSF and 2.5μ gm/ml in the serum using rabbit antisera. Greater sensitivity was achieved when the specimen was concentrated 100-fold. In patients with active coccidioidomycosis, 93 of 96 specimens were positive by CIE and this correlated well with complement-fixation and double immunodiffusion. CIE also has been used for the diagnosis of amebiasis.[36,37] CIE was positive in 90 percent of confirmed cases with amoebic liver abscess and amoebic dysentery. CIE has been found to be useful in the diagnosis of the following parasitic diseases: hydatid disease, fascioliasis, and kala-azar.[38-40] In hydatid disease, serum was tested for antibodies using whole hydatid fluid as antigen while in kala-azar the antigen was prepared from the promastigote form of leishamania donovani. However, especially with fascioliasis, cross-reactions occurred with other parasites when crude antigen was used. Partial purification of the antigen lowered the cross-reactivity.

Numerous modifications of the basic technique of countercurrent immunoelectrophoresis have appeared in the literature. The various modifications are summarized in Table V. Coonrod and Rytel evaluated in great detail the technique of CIE using purified pneumococcal polysaccharide and compared agar matrices and buffers of different pH and ionic strengths.[18] They concluded that a veronal buffer of 0.05 ionic strength and a pH of 8.2 gave the best resolution of precipitin bands. They also reported the CIE sensitivity of the different pneumococcal serotypes using the Statens Seruminstitut antisera. In general, the type-specific antisera proved to be more sensitive than the omniserum which contains antibody against the 82 known pneumococcal serotypes. Pneumococcal types seven and 14 frequently have been noted to give false-negative reactions. Anhalt and Yu described an improved method of detecting pneumococcal types seven and 14 by incorporating a sulfonated derivative of phenylboronic acid to the buffer.[41] This improved the migration abilities of the two types thus increasing the sensitivity.

In a sequential analysis of serum and urine by CIE, Fossieck *et al.* were able to detect the persistence of antigen.[42] In one patient with empyema, CIE was positive up to 32 days. Coonrod and Drennan later reported the kinetics of antigenemia in a sophisticated study of 19 patients with pneumococcal pneumonia.[43] They observed that antigenemia was more common in the lower-numbered serotypes of pneumococcus. They also demonstrated that antigen clearance was dependent upon the appearance of type-specific antibody. Although the concentration of capsular polysaccharide gradually declined during antibiotic therapy, the capsular antigen remained positive in two-thirds of their cases for at least two weeks. Feldman also showed that antibiotic therapy did not significantly alter the presence of antigen during the first 24 hours if the concentration of the antigen exceeded 1μ gm/ml.[44] These studies support the usefulness of CIE in the diagnosis of partially treated infections. Other studies indeed have confirmed the usefulness of CIE in the diagnosis of partially treated infections.[16,19,45]

Several attempts have been made to prognosticate patients with bacterial meningitis using the concentration of antigen in the CSF as a guide.[14,45] Increased amounts of antigen correlated well with poor prognosis. However, Feldman studied the relationship of bacterial concentration and the amount of capsular antigens in the CSF with clinical outcome.[44] He suggested that the concentration of bacteria may be a more important determinant of prognosis.

Although the usefulness of CIE has been well documented, the procedure remains plagued with problems related to sensitivity and specificity. Using a standard amount of antigen, Ingram *et al.* demonstrated differences in the sensitivity and specificity of H. influenzae type b antisera acquired

from different sources.[16] Cross-reacting antigens altered the specificity of the test giving false-positive reactions. Table VI shows the possible cross-reactions between different organisms. They emphasized the cross-reactions between the capsular polysaccharide of H. influenzae type b and the capsular antigen of E. coli Easter strain and pneumococcus type 29. Furthermore, they pointed out possible cross-reactions between the capsular antigens of H. influenzae type b and pneumococcus types 6, 15 and 35. Cross-reactions also have been observed with other organisms.[17,23,39] Bartram *et al.* demonstrated cross-reactions between the pseudomonas antisera and ultra sonic extracts of Proteus sp., K. pneumoniae, E. coli and E. aerogenes.[17] They also mentioned that pseudomonas aeroginosa shared common antigens with S. pneumoniae, E. coli, K. pneumoniae, H. influenzae, achromobacter, vibrio, and aeromonas. Problems related to the sensitivity of the procedure have resulted in false-negative reactions. In a few patients with culture-positive bacterial infections, the CIE gave negative results.[45,46] A recent report showed a lot to lot variation in the sensitivity of a commercially available H. influenzae type b antisera commonly used for CIE.[46]

New developments continually appear in the literature with the purpose of improving the technique and expanding its scope to include other organisms. Recent work on pneumocystis carinii infection appears promising and hopefully will replace invasive procedures such as a lung biopsy.[47] However, the basic technique still requires a great deal of improvement and additional studies need to be developed.

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Table V
Summary of Different CIE Techniques

Reference	Technique	Sensitivity (μgm/ml)
Edwards: *J Immunol* 106:314, 1971	3mA 15 min. room temp.	0.048
Dorff: *Loncet* 1:587 (March) 1971	12-15 V per cm room temp.	no sensitivity stated
Greenwood: *Lancet* 2:519 (Sept) 1971	10mA 45 min. room temp.	0.02
Coonrod: *Lancet* 1:1154 (May) 1972	12-15 V per cm room temp. then cooled to 5°C	0.05
Edwards: *J Lab Clin Med* 80:449, 1972	6mA 30 min room temp.	0.02
Ingram: *J Pediat* 81:1156, 1972	55 V (11mA) 25°C 1½ h 200 V (30mA) 4°C 1½ h	0.03
Fossieck: *J Infect Dis* 127:106, 1973	current? 25 min 37°C	0.05
Coonrod: *J Lob Clin Med* 81:770, 1973	2.5 V per cm 400 V 1 h room temp	0.05
Bartram: *J Lob Clin Med* 83:591, 1974	13.3 V per cm 45 min room temp	no sensitivity stated
Shackelford: *J Pediotr* 85:478, 1974	40mA 30 min room temp then 4°C for 30 min.	0.01 (H. flu) 0.05 (pheumococcus & meningococcus
Nagel: *Ann Intern Med* 82:13, 1975	5.4 V per cm 40 min room temp.	no sensitivity stated
Michaels: *J Pediotr* 88:72, 1976	40-50 mA 45 min room temp.	no sensitivity stated
Lampe: *J Pediotr* 88:557, 1976	24mA 1 h room temp.	no sensitivity stated

Table VI
Cross-Reocting Antigens (CRA)

I. Pneumococcus[1]	K. pneumoniae E. coli H. influenzae type b P. aeroginosa
II. H. influenzae type b	Pneumococcus type 6, 15, 29 and 35 E. coli (Easter strain) S. aureus, S. epidermidis Beta and viridans strep Bacillus sp. Pseudomonas
III. Staphylococci[2,3]	Streptococci S. epidermidis Bacillus sp. Lactobacillus sp. H. influenzae type b
IV. Pseudomonas[4]	S. pneumoniae E. coli K. pneumoniae H. influenzae type b Achromobacter Vibrio Aeromonas Proteus E. aerogenes

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MEDICAL HISTORY

Medical Aspects of the Middlebrook Encampments*

MORRIS H. SAFFRON, M.D., Passaic

Neither of the Middlebrook encampments was notable for unusually severe problems of disease control. Nevertheless these periods were of some importance in the history of the Medical Department, coinciding with developments relating to the continuing search for parity of physicans within the Service. Medical leaders at Middlebrook were William Burnet of Newark and John Cochran of New Brunswick, both early presidents of our Society.

It was toward the end of May, 1777 that George Washington decided to move his troops some ten miles south from the security of Morristown to the slightly more exposed but strategically advantageous area near the Middlebrook. The army then numbered about 8,400 men of whom nearly one-third officially were listed as sick or disabled, but this latter estimate requires an explanation. We know that the Baptist and Presbyterian churches which served as smallpox hospitals had not been released to their congregations, so it is safe to assume that the more seriously ill had been left behind to rejoin their cadres later on. Those who marched or were carried from Westfield to Chatham were suffering from minor ailments or recovering from the effects of the smallpox inoculations recently conducted on a large scale. Smallpox had been and was to remain one of the dreaded scourges of the Continental army throughout the war. It will be recalled that Jenner's discovery was still some twenty years away, and there was always the danger that inoculum obtained directly from the pustule of a patient might incite a serious or even a fatal form of the disease. Despite the supervision of a highly skilled operator—in this case Dr. John Cochran of New Brunswick—one had to anticipate the loss of two or three men of the three hundred usually inoculated as a group. By this time Washington, who came from Virginia where the practice of inoculation was still forbidden by law, had overcome his early misgivings about the procedure and become an enthusiastic convert. Writing from Middlebrook to a brother-in-law he has this to say:

"In general any old woman may inoculate with as much success as the best physician, the whole art lying in keeping the Patient rather low in diet and cool especially in the period of the eruptive fever . . . in general neither Physicians nor physic is necessary except a few purgatives."

It is true that smallpox never posed a serious threat in either of the two Middlebrook encampments—in contrast to Morristown and Valley Forge—yet it should be noted that the isolation hospital at Mendham was not abandoned until the army moved permanently from New Jersey three years later. Washington also mentions a hospital at Basking Ridge, but so far as is known there was no major hospital within the encampment area itself. At Middlebrook during the heat of the summer, the Commander-in-Chief showed a commendable concern for the health of his men. He issued several general orders warning the troops of the necessity of maintaining personal hygiene as well as cabin and camp sanitation, paying particular attention to the importance of a pure water supply. Since many of the enlisted men were farm boys with the most primitive ideas on the subject of waste disposal, his efforts met with only limited success, and at

*Presented at a conference sponsored by the New Jersey Historical Commission and other societies, September 30, 1978, Bound Brook Presbyterian Church.

least one pool became so contaminated as to be declared off limits. The General also pleaded with the pork and beef-loving soldiers to boil rather than broil or roast their meats, discanting on the virtues of vegetables and "salats" in preventing scurvy and other "putrid disorders." He prohibited bathing in the heat of the day or too soon after meals, and encouraged the use of light cottons rather than the flannels to which the men were accustomed. It was during the period of the first Middlebrook encampment that Congress, at Washington's request, considered the plans for establishing a Corps of Invalids which first had been suggested a year earlier by Colonel Lewis Nicola. The partially disabled men of this corps were to be kept in service, but given light duties as hospital attendants. At the same time plans were formulated for pensioning the completely disabled and providing for the families of casualties.

During the early days of the war on Jersey soil, Continental surgeons had treated the enemy wounded in the spirit of compassion called for by the profession, but by the time of Middlebrook I bitterness at Hessian outrages had become so intense that no regimental surgeon could afford to show too much interest in the fate of an injured foe. The true state of affairs becomes clear from the apologetic tone of Washington's letter to Lord William Howe, dated June 10.

"In respect to the attention paid to the sick I am sorry their accommodation was injured in any degree by the misconduct of our surgeons. I heartily join with you in reprobating their proceedings and shall esteem it a favor if you will point out the persons and furnish me with such proof of their guilt as you are possessed of."

One of the most important medical developments at this first Middlebrook encampment related to the long smoldering antagonism between medical personnel of the Line and the Staff of the General Hospital.** Regimental hospitals manned by politically oriented and frequently ill-trained surgeons had existed from the beginning of the war, but gradually the latter had shown marked improvement in competence and reliability. It was at this point that Congress with its usual bumbling interference passed a resolution "that every sick soldier shall be sent to a continental hospital as soon as any other diet or lodging becomes necessary for him, than what is allowed to all soldiers." This lofty idea sounded fine on paper, but in practice it led to many difficulties and reactivated hard feelings. In the first place it humiliated the regimental surgeons by suggesting incompetence; in the second place it raised the spectre of transporting seriously ill men over deeply rutted roads in all kinds of weather; and, finally, it soon resulted in the overcrowding of the so-called general hospitals, usually barns or churchhouses, with patients who might just as well have been treated in camp. As early as June 3rd, it was ordered that regimental surgeons were "not to send any of their sick to hospital without first obtaining a certificate from the surgeon general or one of the deputies for the purpose." Soldiers recuperating in hospitals away from their regiments often became unruly, and the commanding officer nearest to such an installation was commanded to "furnish a good and prudent officer to assist in the government of it as far as keeping the convalescent soldiers in order"; that abuses were to follow this order is indicated by the following:

"Complaints having been made to the Commander-in-chief that some officers, fond of any pretext to get out of the way of their duty, obtrude themselves upon the hospitals without answering any useful end, but rather serving to embarrass and take up room that might be better employed. He orders that not more than one officer attend any hospital unless by application from one of the Directors or Physicians and Surgeons General of the Army. . ."

Ill-feeling was heightened when at the end of June, Major General John Sullivan complained to Washington that some of the sick of his division arbitrarily had been refused admission to a general hospital. This provoked a fiery response from Surgeon General John Cochran which in turn led to a Court of Inquiry, but no further action was taken against Cochran, who I feel certain had Washington's tacit support. This incident, although in itself unpleasant, helped medical officers to attain somewhat later that equality with the Line which had consistently been denied them in the British service.

SECOND ENCAMPMENT

Approximately a year-and-a-half was to pass before Washington's army returned to Middlebrook for the second encampment (November 28, 1778—June 3, 1779) and the medical officers had learned much from their experiences at Brandywine, Germantown, Valley Forge, and Monmouth. Indiscriminate recruiting practices had been abandoned, and only young men in good physical condition and free from obvious disease were encouraged to join the service. As early as November 24th the Flying Hospital had been ordered to the Middlebrook area, and fine huts were erected alongside the Pennsylvania Line at Jockey Hollow. With improvement in food supplies and clothing allotments there was relatively little illness in camp. In a letter to Lafayette, Washington writes glowingly about the advantages of Middlebrook, insisting that the men were "better clad and more healthy than they have been since the formation of the army." In an advertisement to spur enlistments he added:

"The public may depend on it that the greatest exertions shall constantly be made and no care spared to make the sick and wounded comfortable and happy, that all military hospitals of the United States are in excellent order and that the army enjoys a degree of health seldom to be seen or read of."

Undoubtedly the most important factor in the improved health and morale of the troops was the unusual clemency of the weather. Although surgeon's mate James Thacher at first complained about "exposure to cold and storms" in canvas tents, he later admitted, "we have passed a winter remarkably mild and moderate; since January 10th we have had scarcely a fall of snow, or a frost, and no severe weather." By February 1st all the men were in huts, now built according to the recommendations of Washington who blamed slovenly construction practices for much of the illness at Valley Forge. There the huts had been only partially excavated from the earth and the men "had failed to erect births or bunks to keep them off the ground." One benefit of the improved facilities was that scabies, a disease which spread rapidly when men huddled together from the cold, never reached major proportions at Middlebrook. Another helpful factor from the standpoint of health and sanitation was the presence at headquarters of Baron von Steuben, a true martinet who had served as aide-de-camp to Frederick the Great. Known as a stickler for personal and camp cleanliness, he inspected the troops with unusual care insisting on a critical examination of the accommodations being provided for the sick and disabled in camp by the regimental surgeons. General orders now required that each morning an

**Saffron MH: Confrontation in New Jersey. *J Med Soc NJ* 73:1081-1087; 1976.

orderly sergeant list the sick belonging to his company, reporting this to the commanding officer of the regiment, who in turn reported to the surgeon. The orderly sergeant was also to accompany the latter on his rounds, distribute medicine and carry out all instructions. On one occasion he was told to assign "a proportionate number of women who were to be sent to the hospitals at Black River and Mendham to attend the sick as nurses."However it was at New Brunswick and its immediate environs that there seems to have been the greatest concentration of medical beds, for we have references to several line officers who were assigned to monthly tours of duty supervising the hospitals in that area. Washington also showed concern over the possibility of an enemy attack on outlying hospitals, and on one holiday ordered increased protection for them. Undoubtedly Middlebrook surgeons saw isolated cases of the usual camp diseases such as dysentery, spotted or typhus fever, remittent fever or malaria and putrid fever or meningitis, but the generally healthful conditions at camp may best be inferred from this letter by the conscientious Surgeon General Cochran, whose wife was lying desperately ill at Livingston Manor. Writing for a leave of absence to Dr. James McHenry, one of Washington's "family," he says:

"I flatter myself my absence will be attended with no injury to the service. I suppose that there are general hospitals established near your quarters for the reception of the regimental sick and Draper will attend to seeing them conveyed thither at proper times and seasons and that the regimental sick are provided with the usual stores, the same as last year."

Apparently Dr. George Draper was able to carry on alone during his superior's absence, receiving reports and issuing orders and supplies from the medical headquarters at Mr. Michael Field's in Bound Brook. From camp Washington now sent Congress a critique of the Medical Department, urging that there be one medical director with undisputed authority to move surgeons from one District to another as needed; he also suggested "more latitude for the regimental surgeons" who would be placed under the authority of the more sympathetic Surgeon General of the Flying Hospital (Cochran) rather than the Director General (Shippen). Things were so restful in camp that in February a course of lectures on anatomy and surgery, supervised by Dr. William Brown of Virginia, was announced. They were to be attended by all junior surgeons. I am inclined to think that these lectures well may represent the first attempt at formal medical instruction in the State of New Jersey.

It is generally assumed that there was a great deal of sympathy for the sick and wounded even from those who had loyalist leanings. That this was not always the case is shown by this letter by Surgeon Barnabas Binney to General Nathanael Greene. From his station at Somerset Court House (Millstone) he writes on May 18, 1779:

"The two churches and court house of which they (the townspeople) have legal possession being crowded to a degree dangerous to the health of the wounded (the writer) has applied to the magistrates for the neighboring barns to accommodate the convalescents. In answer they threaten to imprison the first who shall prostitute a barn for the sake of sick soldiers. What's to be done?"

These barns were soon numbered among the total of forty-one which eventually were adapted for hospital use in the Middlebrook area. The surgery performed in such quarters, though not worse than the British, and certainly the equal of the French and German according to James Tilton, beggars the imagination. If the poor victim could survive an amputation—no matter how swiftly and skillfully performed—with no other anesthetic than a tumbler of gin or a lead bullet to bite on, then he had one chance out of three of dying from the infection which almost invariably followed. Even vinegar, the antiseptic most constantly used, was in short supply at Middlebrook. Closing of wounds was performed by the use of adhesive strips or sutures made of shoemaker's wax ends, left long and removed in two or three days. To make matters worse Dr. James Hutchinson reports that there was such a scarcity of lancets at Middlebrook that some of the regimental surgeons and even those at the Flying Hospital had to do without them. It is estimated that one surgeon had to supervise the health of 1,000 men and that one male nurse was expected to take care of at least ten patients. Nevertheless there were only one hundred deaths reported at Middlebrook from February 20 to May 29 as compared with the 2,100 recorded at Valley Forge over a slightly longer period.

When Washington readied himself to move his army to the Hudson he ordered that the sick from Somerset and those at camp be moved to the well-constructed huts at Pluckemin which the artillery was about to vacate, all of this to be done gradually and carefully with the assistance of the Quartermaster. Finally, it should be noted that although desertions at Middlebrook were remarkably few, there is good reason to believe that there as elsewhere much of the illness was due to a longing to be at home, free from the ever-present threat of hunger and cold. This nostalgia often could be cured by the raillery of older soldiers, strenuous exercise, or the prospect of battle, but as General Schuyler once aptly put it:

"Of all the specifics ever invented there is none so efficacious as a discharge, for as soon as their faces turn homeward nine out of ten are cured."

Medical Officers Known To Have Served At Middlebrook

Baldwin, Cornelius	Surgeon, 2nd Essex Regiment
Barber, Thomas	Surgeon, 1st Monmouth Regiment
Barnet, Oliver	Surgeon, 4th Hunterdon Regiment
Binney, Barnabas	Surgeon, General Hospital
Blachly, Ebenezer	Surgeon, Pennsylvania Line
Bloomfield, Moses	Surgeon, General Hospital
Brown, William	Physician and Surgeon General
Burnet, William	Surgeon General, Eastern Department
Burnet, William, Jr.	Surgeon, General Hospital
Campfield, Jabez	Surgeon, Spencer's Regiment
Cochran, John	Surgeon General, Flying Hospital
Darcy, John	Surgeon's mate, Spencer's Regiment
Draper, George	Surgeon, General Hospital
Dunham, Lewis	Surgeon's mate, 3rd N.J. Regiment
Elmer, Ebenezer	Surgeon, General Hospital
Harris, Jacob	Surgeon's mate, 4th Battalion
Henry, Robert L.	Surgeon's mate General Hospital
Hutchinson, James	Surgeon, Pennsylvania Line
Little, —	Surgeon's mate, Massachusetts
Loring, Ephraim	Surgeon's mate, 3rd Battalion
McCarter, Chester	Surgeon, 4th Pennsylvania Regiment
McKenzie, Samuel	Surgeon, Pennsylvania Line
Thacher, James	Surgeon, 1st Virginia Regiment
Wilson, Louis F.	Surgeon, General Hospital

Alcohol-Nutrition Interaction

CHARLES S. LIEBER, M.D., East Bronx, New York*

Alcohol and nutrition interact at many levels. Ethanol directly may alter the level of nutrient intake through its effect on appetite, its displacement of food in the diet, or its effect on the gastrointestinal tract resulting in disturbances of digestion and absorption. Through its effect on various organs, especially the liver, ethanol may alter the transport, activation, catabolism, utilization, and storage of nutrients. As a result, alcoholism remains one of the major causes of nutritional deficiencies in our society. Ethanol is directly toxic to many body tissues and this effect may be worsened by existing nutritional deficiencies. Thus, because of its widespread use and multiple effects, ethanol has a major impact on overall nutritional status.

ALCOHOL, MALNUTRITION, AND LIVER INJURY

The question of the respective roles of alcohol and malnutrition in the pathogenesis of liver disease seen in the alcoholic (fatty liver, alcoholic hepatitis, and cirrhosis) is of utmost importance for the prevention and treatment of the disease. Due to the unreliability of accurate nutritional evaluation and the long-time course of pathogenesis, resolving this question has been exceedingly difficult.

Malnutrition has been proposed as the predominant factor in liver injury for several reasons. First, each gram of ethanol provides 7.1 calories, which means that 20 ounces (or 586 ml) of an 86 proof (43% v/v) beverage represents about 1500 calories or 1/2 to 2/3 of the normal daily caloric requirement. Therefore, the alcoholic has a much reduced demand for food to fulfill his caloric needs. Since alcoholic beverages do not contain significant amounts of protein, vitamins and minerals, the intake of these nutrients readily may become borderline or insufficient.

Economic factors also may reduce the consumption of nutrient-rich food by the alcoholic. In addition to acting as "empty" calories, alcohol can result in malnutrition by interfering with the normal processes of food digestion and absorption.[1]

Studies with rodents show that they develop liver disease when fed severely deficient diets even in the absence of alcohol. Researchers extrapolated this to humans and postulated that (1) liver disease is due solely to nutritional deficiencies; therefore, in an adequate diet, alcohol adds only calories and (2) alcohol is no more toxic than a similar caloric load provided by starch or fat.[2]

This opinion prevailed despite statistical evidence gathered both in France[3] and Germany[4], which indicated that the incidence of liver disease correlated with the amount of alcohol consumed rather than with deficiencies in the diet.

A major challenge to the concept of the exclusively nutritional origin of alcoholic liver disease arose from an improvement of the method of alcohol feeding to experimental animals. When the conventional alcohol feeding procedure is used—ethanol given as part of the drinking water—rats usually refuse to take a sufficient amount of ethanol to develop liver injury if the diet is adequate.

This was counteracted by the introduction of the new technique of feeding ethanol as part of a nutritionally adequate, totally liquid diet[5-7]. With this procedure, ethanol intake was sufficient to produce a fatty liver despite an adequate diet. This technique now is adopted widely for the study of the pathogenesis of the fatty liver in the rat. In addition to the fatty liver, ethanol dependence developed in these rats as witnessed by typical withdrawal seizures after cessation of alcohol intake[8].

Having established an etiologic role for ethanol in the pathogenesis of the experimental fatty liver, the question of its importance for human pathology remained. To determine whether ingestion of alcohol in amounts comparable to those consumed by chronic alcoholics is capable of injuring the liver even in the absence of dietary deficiencies, volunteers (with or without a history of alcoholism) were given a variety of nondeficient diets under metabolic ward conditions with ethanol either as a supplement to the diet or as an isocaloric substitution for carbohydrates[5,6]. In all these individuals, ethanol administration resulted in fatty liver development. This was evident by both morphologic examination and direct measurement of the lipid content of the liver biopsies, which revealed a rise in triglyceride concentration up to 15 fold.

The etiological role of alcohol per se (in the absence of dietary deficiency) in the pathogenesis of alcoholic liver injury now has been extended from the fatty liver to the full spectrum of liver disease including cirrhosis. This was achieved by using an experimental model developed in the baboon[9]. In these experimental animals, the sequential de-

*Reprinted with permission of *Contemporary Nutrition* 3:9 (September) 1978, a newsletter from the Nutrition Department of General Mills, Inc., Minneapolis. Dr. Lieber is affiliated with the Alcohol Research Center, Veterans Administration, 130 West Kingsbridge Road, Bronx, New York 10468, and may be addressed there.

velopment of all the liver lesions seen in the human alcoholic was reproduced: 23 baboons fed ethanol all developed fatty livers; five progressed to mild hepatitis and six had cirrhosis. Maintenance of a nutritionally adequate regimen despite the intake of ethanol (50 percent of total calories) was achieved by incorporation of the ethanol in a totally liquid diet. On ethanol withdrawal, signs of physical dependence, such as seizures and tremors, developed.

Ultra-structural changes of mitochondria and the endoplasmic reticulum were already present at the fatty liver stage and persisted throughout the cirrhosis. The lesions were similar to those observed in alcoholics and differed from the alterations produced by choline and protein deficiencies. At the fatty liver stage, some "adaptive" increases in the activity of microsomal enzymes were observed but these tended to disappear with the development of hepatitis and cirrhosis. Fat accumulation was also much more pronounced in the animals with hepatitis as compared with those with simple fatty liver (an 18-fold, compared to three to four-fold, increase in liver triglycerides).

The demonstration that these lesions can develop despite an adequate diet indicates that in addition to correcting nutritional status, controlling alcohol intake is mandatory for the management of patients with alcoholic liver injury. Ethanol per se must be considered a direct etiological agent in the pathogenesis of alcoholic liver injury, independent of dietary factors[10]. However, this does not mean that dietary factors may not contribute to or worsen the alcohol effect. No related human studies are available.

NUTRITIONAL VALUE OF ALCOHOLIC BEVERAGES

Ethanol liberates 7.1 Kcal./gram when combusted but does not provide equivalent caloric food value when compared to carbohydrate. Gain in body weight is significantly lower with ethanol than with isocaloric amounts of carbohydrate.[11]. Also, isocaloric substitution of ethanol for carbohydrate as 50 percent of total calories in a balanced diet results in a weight loss[11]. A possible reason for this is that chronic alcohol intake increases the energy requirements of the body. If this were the case, it should be reflected in a higher rate of oxygen consumption. It was, indeed, found in rats fed alcohol as part of their totally liquid diet that oxygen consumption was slightly but significantly higher than that of animals pair-fed the isocaloric diet substituting carbohydrate for ethanol.[12]

A possible mechanism that could account for an inefficient use of ethanol calories involves the energy wastage secondary to an induction of liver microsomal pathways.

Another theory is an increased utilization of ATP by the sodium-potassium activated ATPase after chronic ethanol feeding. Israel *et al.*[13] reported that in liver slices, ouabain, an inhibitor of the sodium-potassium activitated ATPase, completely blocked the extra ethanol metabolism elicited by chronic ethanol treatment.

The theory that increased utilization of ATP by the sodium-potassium activated ATPase (and the resulting lowering of the phosphorylation potential) is responsible for the metabolic adaption that occurs in rats after chronic ethanol treatment and that a situation develops that is very similar to that found after administration of thyroid hormones or epinephrine is intriguing. Certainly, the influence of ethanol consumption on hormonal actions deserves further investigation. To date, there have been few studies performed to confirm these observations.

Under the conditions used by Israel *et al.*[13] ethanol consumption did not result in liver changes comparable to those seen in human alcoholic liver injury. For instance, no fatty liver was observed. Under conditions that mimic the clinical situation with development of fatty liver, chronic ethanol consumption was not found to be associated with increased ATPase activity[14] and the increase in the rates of ethanol metabolism after chronic ethanol consumption could not be abolished by ouabain[15]. This indicates that the theory of enhanced ATPase activity may not be applicable to the situation that normally prevails after chronic alcohol consumption.

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DOCTOR'S NOTEBOOK

Trustees' Minutes

Regular meetings of the Board of Trustees were held on Sundays, February 25, and on March 18, 1979, at the Executive Offices in Lawrenceville. Detailed minutes are on file with the secretary of your county medical society. A summary of significant actions follows:

February 25, 1979

Southern New Jersey Medical School . . . Noted that President Krueger had presented a paper before the Department of Higher Education's Ad Hoc Committee on Medical Education in Southern New Jersey and participated in a question and answer session regarding MSNJ's position on the Southern New Jersey Medical School Program. A decision will be rendered some time in April.

American Academy of Pediatrics . . . Noted that there was dissatisfaction among the pediatricians concerning a change from Classification I to II by the Medical Inter-Insurance Exchange of New Jersey. No increase in rate resulted, but it was felt that the change was abrupt and more sensitivity should be displayed before such changes are made. A communication will be sent to MIIE expressing these thoughts.

Procedure for Recommendation for AMA Appointments . . . Noted that a communication had been received from the AMA detailing the procedure to be followed for appointment to certain AMA committees, and approved the following recommendation:

That the Board of Trustees approve the submission of the name of Bernard A. Rineberg, M.D., as nominee for appointment to the AMA Review Committee for Orthopaedic Surgery.

Excessive Fees for Professional Services —Proposed N.J.A.C. 13:35-6.12 . . . Approved the following recommendation of the Executive Committee:

That a communication be sent to the State Board of Medical Examiners suggesting that the State Board consider utilizing the MSNJ judicial mechanism in any regulatory system developed to address the question of excessive fees for professional services.

Requirements for Issuing a Prescription Establishing a Requirement of Notice to the Public of Generic Drug Availability— Proposed Amendment to N.J.A.C. 13:35-6.6 . . . Approved the following recommendation of the Executive Committee:

That a communication be directed to the State Board of Medical Examiners indicating that MSNJ will assist in the implementation of the rule on generic drug availability. It is to be noted that the formulary required by the statute has not as yet been adopted fully by the Department of Health.

Ad Hoc Committee on Emergency Medical Services Regionalization Proposal . . . Approved the following recommendation of the Executive Committee:

That the Board approve the formation of an ad hoc committee, to be chaired by Alfred A. Alessi, M.D., to address the EMS Regionalization Proposal of the State Department of Health.

Note: The following will be requested to serve on the committee: Doctors R. Winfield Betts, William A. Dwyer, Jr., Paul J. Hirsch, Jack R. Karel, Morton W. Leach, Henry R. Liss, Daniel J. O'Regan, Benjamin F. Rush, Jr., Rudolph E. Schwaeble, and Michael D. Yablonski. William F. Minogue, M.D., Medical Director of the EMS Project, and Mr. Louis Scibetta, of the New Jersey Hospital Association, will be invited to participate.

Conference on Urban Health and Economics . . . Approved the following recommendation of the Executive Committee:

That cosponsorship of the Conference on Urban Health and Economics be declined.

Note: It was the opinion of the Executive Committee that the program for the conference had been completed when the invitation was extended, and that MSNJ would have no input. MSNJ will offer to publicize the meeting and Mr. Joseph Lucci or a member of the staff will be assigned to attend.

Attendance of Secretary at AMA House of Delegates . . . Approved the following recommendation of the Executive Committee:

That the Secretary be authorized to attend the Interim and Annual Meetings of the AMA House of Delegates as is customary for the President, President-Elect, AMA Delegates and Alternate Delegates, and the Executive Director.

CMDNJ . . . Received a report from the President of the College of Medicine and Dentistry of New Jersey which highlighted the following points:

1. **Governor's Budget** . . . Proposed an appropriation of $62.7 million to CMDNJ for fiscal year 1980, up from $59.23 million this year.

2. **Tuition** . . . The Governor's budget recommendation assumed tuition increases averaging 23.2 percent at all CMDNJ schools, and 25 percent (or $1,000 more per year) at CMDNJ medical and dental schools.

3. **Graduate Medical Education Council of New Jersey** . . . Foreign medical graduates in New Jersey hospital residency programs dropped from 80 to 55 percent in 1978. Of 920 U.S. citizens in New Jersey residencies, 359 were natives of the state, and 258 were CMDNJ graduates. The percentage of first year primary-care residents was 54 in 1978 (legislative target by that time was 35 percent). In May the Council will announce one-year grants totaling $240,000 to develop additional hospital-based residencies throughout the state.

4. **South Jersey Medical Education Program** . . . The College supports the CMDNJ Trustees' proposal which would increase CMDNJ-Rutgers Medi-

cal School to 140 students per class with 108 per class pursuing third and fourth years at affiliated hospitals in central Jersey and 32 per class pursuing the clinical years in south Jersey, and the growth of CMDNJ-Osteopathic School to 48 students per class and the school's removal to Camden. Under such a plan the College and the State would fulfill commitments under Senate Bill 1540, mandating creation and maintenance of osteopathic and allopathic medical education programs in South Jersey.

5. **Rutgers Medical School** has asked permission to increase the size of its third-year class to 108 students, citing the school's increased capacity in clinical education through its affiliation with Middlesex General Hospital and Cooper Medical Center. Twelve full-time RMS faculty are at Cooper and seven at Middlesex.

6. **Camden Area Health Education Center** . . . Mervyn A. Landay, D.D.S., has been named program director for the Camden Area Health Education Center which will be developed with joint funding from the federal government and the College.

7. **College Hospital** . . . College Hospital opened on January 20 and its emergency room began functioning two nights later.

8. **Conjoint Cancer Center** . . . The State Health Coordinating Council recommended approval of the creation of the Conjoint Center for Radiation Oncology and the issuance of a certificate of need for a 20-24 MEV linear accelerator at the College Hospital.

9. **New Jersey Medical School Department** . . . CMDNJ Trustees appointed Carroll Leevy, M.D., Chairman of the CMDNJ-New Jersey Medical School's Department of Medicine and Physician-in-Chief of the College Hospital.

New Jersey Hospital Association . . . Received as informative the report from NJHA which congratulated MSNJ on moving to our new headquarters. Mention was made that NJHA still is concerned that the Diagnostic Related Group program has not proved itself and should continue to be run as an experiment for at least a year. Attention was called to the position by the Carter administration seeking legislation to set rates for physicians and hospitals. Concerning podiatrists, NJHA will welcome comments on the issue of granting privileges to podiatrists in hospitals.

Medical Society of New Jersey Student Association . . . Noted that present membership stands at 187 students, the new liaison representative is to be Marc Rappaport, and approved a recommendation for financial assistance to two students to attend the annual meeting of the American Medical Student Association in Denver in March.

. . .Approved a recommendation that the Board of Trustees suggest the ratio of one student per 100 student-members to represent the MSNJ Student Association at the AMA Annual and Interim Meetings, MSNJ's Annual Meeting, and the AMSA National Convention.

Psychotropic Medication . . .Referred back to the Council on Mental Health a recommendation from that Council that the Board of Trustees request the Commissioner of Human Services to withdraw Administrative Bulletin 78-6, which lists guidelines for administration of psychotropic medication, with the request that the proposed guidelines be discussed with Dr. Julia R. Allen of the Department of Human Services.

Long Range Planning and Development . . . Adopted the following statement of goals and approved the recommendation of the Committee on Long Range Planning and Development:

Goals

The Committee believes that if the Society is to be effective in a long-range planning and development effort it must, of necessity, look toward the goals it has set, i.e., a viable, effective association to advance the interests of the practitioners of medicine through scientific, educational, socio-economic, and legislative programs; evaluate the barriers and the means to achieving the desired goals; and implement the mechanisms to carry through to completion.

Recommendation

That at the Annual Meeting of the Medical Society of New Jersey, the chairmen of the reference committees be charged with making a list of problems that become evident during their meetings; and that such lists be presented to the President and to the Board of Trustees for referral to the Committee on Long Range Planning and Development and other appropriate committees for study and recommendation during the next year. The Committee will, of course, continue to study and explore areas as it deems advisable.

. . .Voted to adopt the charges to the Committee as developed by the Council on Long Range Planning and Development of the AMA which are:

1. To study and make recommendations concerning the long-range objectives of the association.

2. To study and make recommendations concerning the projected resources, programs and organizational structure by which the association attempts to reach its long-range objectives in the above.

3. To serve as a focal point for the planning activities of the association and to stimulate and evaluate planning activities throughout the organization.

4. To study, or cause to be studied, the future environment in which medicine and the association must function, collect relevant data and transmit interpretations of these studies and data to the Board of Trustees for distribution to decision-making centers throughout the association, and submit reports to the House of Delegates at appropriate times.

Specialty Society Representation in House of Delegates . . . Referred to the Committee on Revision of Constitution and Bylaws for study and report the following recommendation:

That the Bylaws of the Medical Society of New Jersey be amended so that each of the specialty societies in New Jersey (affiliated with national specialty societies recognized by the AMA) shall appoint or elect a delegate to the House of Delegates of the Medical Society of New Jersey. Such delegates shall have full voting powers and responsibilities.

Regionalization of Emergency Medical Services . . . Noted that the Health Care Administration Board directed that revised guidelines for the regionalization of emergency medical services be published in the *New Jersey Register.*

Cooperation in Filling Third Party Forms . . .Noted in a report from Dr. Anthony P. DeSpirito, MSNJ's representative at the January 10 meeting of the State Board of Medical Examiners, that a communication from Assemblyman Walter J. Kozloski—concerning payment of physician's bills—was discussed. (Assemblyman Kozloski had had difficulty in obtaining insurance forms from an anesthesiologist.)

. . .Directed that a communication be sent to Assemblyman Kozloski offering assistance, if he would supply the name of the physician, by reminding that physician of his responsibility in completing third party forms.

Critical Care Medicine . . . Directed that the recently formed New Jersey Society

of Critical Care Medicine be informed that it is not eligible for component society status since only county medical societies are granted such status.

. . .Agreed that the group should be encouraged to participate with other specialty societies in the scientific sessions at the Annual Meeting through communication with the Committee on Annual Meeting or the Convention Manager.

Note: Purpose of the newly formed organization is to increase awareness of physicians in aspects of critical care medicine as well as to educate members in the newest techniques in this field. Membership is open to physicians, Ph.Ds, scientists, and paramedical personnel (such as nurses, respiratory therapists, and others).

Child Health Conference/Well-Baby Clinics . . . Referred to the Special Committee on Child Health, for consideration and report, a communication from the Union County Medical Society which refers to a letter from Dr. Joseph Boylan, Chief of Pediatrics at Overlook Hospital, concerning the operation of Child Health Conferences/Well-Baby Clinics at various locales in New Jersey, which offer services without charge to all residents of all communities. Dr. Boylan feels that this action is in direct conflict to the private practice of medicine and with the concept that infants and children require continuous and comprehensive care, not merely sporadic care for an illness.

Guidelines for Hypertension Control . . . Named Arthur Bernstein, M.D., to represent MSNJ in the formation of a task force to develop statewide guidelines for hypertension control, requested of the American Heart Association, New Jersey affiliate, by the Office of Chronic Diseases of the Department of Health.

Nominating Committee . . .Directed that the Nominating Committee be asked to reassemble to reconsider the nominees recommended for AMA Alternate Delegates, and that the nominating delegates be provided with the pertinent portion of *Sturgis,* applicable to the Nominating Committee, and directed further that Dr. Mineur be present in the capacity of parliamentarian.*

Note: The action is in response to a communication from the President of the Hudson County Medical Society, questioning the parliamentary propriety of the procedure of nomination at the meeting of the Nominating Committee held on February 14.

. . .Directed that the Committee on Revision of Constitution and Bylaws be made aware of the problems of the Nominating Committee in interpreting the rules of *Sturgis,* and that an evaluation be made of the functions of the Speaker of the House between meetings.

*Nominating Committee report was amended by designating Charles L. Cunniff, M.D. (Hudson) as nominee for AMA Alternate Delegate in place of Edward A. Schauer, M.D. (Monmouth) who was named at the February 14th meeting of the Committee, and by nominating George L. Triebenbacker, M.D. (Ocean) for membership on the Mental Health Council from the 4th District.

March 18, 1979

Southern New Jersey Medical School . . . Considered remarks by Benjamin Cohen, D.O., who made the following points favoring construction of the proposed basic science facility in South Jersey.

1. Rutgers Medical School was designed to accommodate 80 students; enrollment exceeds 80 students. Space is limited; laboratories and faculty rooms are crowded, and there is no space for expansion;

2. Present conditions make it difficult to attract top-level faculty—creating an academic void;

3. Clinical studies begin in the first year and the distance between clinical and basic science facilities creates problems for students and faculty. Students must be transported by bus for patient exposure.

4. It is not intended to duplicate tertiary care facilities; DO's will be trained in allopathic facilities and no new competitive facilities will be built.

Dr. Bergen indicated that regardless of where osteopathic students are trained for basic science, allopathic students will be rotated through South Jersey facilities for clinical training as well as through Newark and Rutgers. It has been shown that a certain percentage of students remain in the area where they receive their clinical training.

. . . Concurred with the Executive Committee that complete knowledge of this matter is of paramount importance to all physicians and residents of New Jersey and that the House of Delegates should address the issue at the annual meeting in May.

. . . Directed that no change in its position be made ". . . that for the present there should be no establishment of a basic science facility in Southern New Jersey"; but that this position be considered by the House of Delegates along with background materials from Camden County Medical Society, the New Jersey Association of Osteopathic Physicians and Surgeons, and a position paper from CMDNJ (if prepared).

Note: The above actions were precipitated by a meeting on March 14, 1979 of the Executive Committee of MSNJ and the Executive Committee of the New Jersey Association of Osteopathic Physicians and Surgeons.

New Professional Liability Supreme Court Subpanels . . . Noted that these subpanels are functioning and have tried 12 cases to date, with unanimous decisions in each case—ten in favor of the defendant; two in favor of the plaintiff.

Medical Directors of Long-Term Care Institutions . . . Noted that an organization of medical directors of long-term care institutions is being formed, that advance monies were requested by David Eckstein, M.D., Chairman of MSNJ's Committee on the Chronically Ill and Aging, and approved the following recommendation:

That the Board authorize start-up funds in the amount of $500 for the organization of medical directors of long-term care institutions.

Use of MSNJ Headquarters by State Board of Medical Examiners . . . Agreed to permit the State Board of Medical Examiners to use the MSNJ facility on June 12, 13, and 14, 1979, at no cost, providing no State facility is available, on a one-time basis.

New Jersey Hospital Association . . . Received as informative, in the absence of a formal report, the following item:

Inner-City Hospitals hope to effect an interim program under Bill S-446. Mon-

ey is available to hospitals caring for large numbers of indigent patients. The New Jersey Hospital Association is working with the New Jersey Department of Health and Blue Cross Association on this matter.

Medical Society of New Jersey Student Association . . . Considered remarks by Marty Skoczyles, member of MSNJ's Student Association, concerning tuition increases and student loan limitations, and agreed that:

1. Tuition increases should be kept in line with President Carter's anti-inflation measures.
2. The limitation on student loans should be expanded.
3. MSNJ should support the proposed task force and give assistance as needed. (Task force is proposed to investigate the possibility of expanding the current limitation on student 7 percent deferrable loans.)
4. Support favorable consideration of Assembly Bill 263 which would enable the State to loan funds to New Jersey medical and dental students and includes a program for loan redemption.

MSNJ Executive Committee Meeting with Representatives of NJHA . . . Received as informative a report from the MSNJ's Executive Committee meeting with the New Jersey Hospital Association's Executive Committee on the following items:

1. *Development of Hospices* . . . Discussed detailed presentation of the administrator of the hospice in Boonton, funded by a National Cancer Institute grant. Until cost data are available, no formal push should be made to expand inpatient plans.

Note: Resolution #2 (MSNJ, 1978) calls for MSNJ to encourage this concept's development.

2. *Diagnostic Related Group Project* . . . Discussed the fact that the State Department of Health is working toward an implementation date of September 1979 for this concept; despite the fact that the project study phase is still out to bid, not yet contracted for.

. . . Agreed that both organizations should continue to support the study, but indicate clearly their resistance to impressing the system before the concept has been proved.

Note: In January of 1979 MSNJ forwarded a letter to the New Jersey Commissioner of Health concerning the status of the DRG Project, requesting release of information, with sufficient time for study and discussion at a public session before a mandatory system is established. The Commissioner's reply was confusing, and MSNJ will attempt to schedule a meeting with the Commissioner and the joint Executive Committees of MSNJ and NJHA.

MSNJ Executive Committee Meeting with the Committee on Medicaid . . . Received as informative a report of a joint meeting of MSNJ's Executive Committee and the Committee on Medicaid, which covered the following items and took actions as indicated:

1. *Medicaid Questionnaire* . . . Noted that the Committee on Medicaid requested permission to conduct a survey of the membership to obtain data on the reaction of physicians and their patients to audit by Medicaid officials.

. . . Approved the following recommendation:

That the Committee on Medicaid be authorized to conduct a membership survey.

2. *Medicaid Resolution* . . . Considered the Committee on Medicaid's dissatisfaction with lack of progress on negotiations with the State of New Jersey to upgrade the Medicaid program. Progress report stated that MSNJ has submitted statistical data to the State indicating that the program would benefit from an increase in level of reimbursement to physicians for office utilization of procedures. MSNJ suggests that the Division of Medical Assistance should encourage its recipients to utilize the private physician's office rather than hospital clinic or outpatient departments. There are indications that the government will reallocate funds providing more rational fee levels for physicians; however, hospitals would resist such a transfer. Component societies are to receive copies of the statistical materials submitted to the Governor's staff.

. . . Agreed to continue efforts to increase the level of reimbursement to physicians for office visits and procedures through appropriate channels of the legislature and the executive branch of the State government.

Note: The Medicaid Committee had requested the Board to retain legal counsel to file suit against the Division of Medical Assistance and the Department of Human Services to force a transfer of monies from outpatient department services to private physician services. Request was disapproved after statement by Mr. Maressa that such litigation hardly could succeed.

MSNJ Executive Committee Meeting with New Jersey Foundation for Health Care Evaluation . . . Received as informative a report from the Executive Committee and its meeting with representatives of the New Jersey Foundation for Health Care Evaluation which considered the Foundation's Statewide IPA/HMO Feasibility Study. Their findings indicate that development of a statewide HMO, licensed as a New Jersey entity (contracting with the various IPAs) would be the best path for MSNJ to pursue. The statewide HMO would have to be organized separately and would require a certificate of need. The Executive Committee advised the NJFHCE to channel its efforts in that direction and to proceed with their study and report.

Society of Anesthesiologists . . . Referred back to the Council on Medical Services the matter of inequities in the area of economics experienced by members of the New Jersey State Society of Anesthesiologists with instructions to consider the overall problem; not to concern itself with the current dispute between NJSSA and Blue Shield.

. . . Requested that a mechanism be developed which MSNJ can offer to specialty societies as an alternative when disputes arise between them and third parties, along with recommendations for the Society's role in negotiating amicable solutions.

Accreditation of Newton Hospital . . . Received a report from the Committee on Medical Education concerning action of the Liaison Committee on Continuing Medical Education Review in deferring accreditation of Newton Hospital pending receipt of additional information on the objectives and curriculum of its program. In view of the fact that the National Council of State Committees on Continuing Medical Education has been questioning the function of LCCME and its accreditation decisions, the Board approved the following recommendation:

That the Board of Trustees send a communication to the Secretary of the LCCME Review Committee protesting its procedure of reviewing material that already has been

evaluated by MSNJ. (LCCME has accredited MSNJ to review hospitals. Therefore, this is an unnecessary duplication of an ongoing effective program.)

CME Delinquent Members . . . Noted that 89 physicians in 1977 and 148 in 1978 have not completed their requirements for continuing medical education, despite letters reminding them of their failure to comply. The Committee was instructed to terminate membership of all physicians who have not met CME requirements within 18 months of their due date.

Amphetamines . . . Considered Union County's resolution to the State Board of Medical Examiners concerning implementation of NJAC 13:35-6.16, which the county claims is unduly restrictive upon the judgment of the treating physician, in his customary practice of medicine.

. . . Indicated its willingness to intervene in Union County's behalf if evidence can be supplied documenting specific incidents where the ruling had imposed undue hardship on the physician's medical judgment.

CMDNJ Notes*

Stanley S. Bergen, Jr., M.D.
President

(This month's column was prepared by Edward J. Cafruny, M.D., Ph.D., dean of the CMDNJ-Graduate School of Biomedical Sciences)

Medical practice is changing constantly. Each year brings new procedures, new drugs, and new information. Much of the clinical and most of the basic science information is required through research conducted in medical schools.

Although basic research excites few people other than those who do it or delve into its mysteries, it is responsible for virtually all major advances in medical practice. Typically, a basic science project begins with a question. Whether the answer has any applied value or not may not be apparent until many years later. For example, research on H_2 receptors did not yield anything of value to clinical medicine for decades, until the H_2 blocker, cimetidine, was discovered. Studies on the renin-angiotensin system, which began in 1898, only now are beginning to reach fruition in the form of a new drug, a converting enzyme inhibitor, which in early clinical trials has been remarkably effective as an antihypertensive (see Brunner *et al: Ann Int Med* 90: 1923, 1979).

The positive impact of basic research on medical practice—the inevitable improvements and advances—is not always perceived. Too many people think of a medical school merely as a place where students learn how to be doctors. Many do not know that all developed medical schools in the United States also train medical scientists who conduct original research during the latter part of their training. This second group of students in our medical schools contributes immensely to medical science and practice. Through research they help to generate the teaching material of the future, set the intellectual tone for the institution, and enrich the biomedical literature.

At the College of Medicine and Dentistry of New Jersey, students in basic science attend the Graduate School of Biomedical Sciences four to six years postbaccalaureate. On completion of their work, they are awarded the Ph.D. degree in one of six areas of study (anatomy, biochemistry, microbiology, pathology, pharmacology, physiology).

New programs in neuroscience and in medical genetics are planned and probably will be offered in the near future.

Admission to the CMDNJ-Graduate School of Biomedical Sciences is highly competitive. Requirements include evidence of superior undergraduate achievement, especially in biology, chemistry, physics, and mathematics. Most of the first two years of training is devoted to courses in the various departments of the school. After completion of course requirements and a final qualifying examination, a student is admitted to candidacy for the Ph.D. degree. The next two years or more are spent developing a dissertation which is based on original research performed in a laboratory in collaboration with a faculty member.

There are presently 63 full-time resident (from New Jersey) and four nonresident students enrolled in the graduate school. Most receive a stipend and financial aid to defray the cost of tuition.

Most of the graduates join a medical school faculty after two years of postdoctoral training at another school. Some choose to work as scientists in government or industry.

Report from the Foundation

Daniel J. O'Regan, M.D.
Medical Director

Analysis of data pertaining to medical care is carried on by many agencies for various purposes. PSROs require utilization details. PSROs and JCAH require Medical Care Evaluation studies. Planning agencies use statistics on patterns of use. Third party payers need information for payment purposes. In New Jersey, the Department of Health collects billing data and medical abstracts on *all* patients. This information will be used to establish prospective reimbursement policies; a study is currently under way in 22 hospitals, using the DRG (Diagnosis-Related Group) classification.

The information which finds its way into the data bank is derived from the medical abstract. The abstract is prepared from the information found in the patient's chart. The chart of a hospital encounter should reflect accurately what occurred during the hospital stay. The reasons for admission, pertinent medical information, therapeutic and diagnostic procedures, disposition and condition on discharge—these are some of the essentials of a good summary.

The *Face Sheet* of the hospital chart is a prime source of information. Face sheet entries often are transmitted directly to the data-using agencies. How the face sheet is filled out can make a great difference in the subsequent stages of information flow. The method of filling in face sheets has changed several times in the thirty years since your reporter's graduation. PSROs refer to the *"Principal Diagnosis,"* which means that condition chiefly responsible for the admission of the patient to the hospital. The *"Principal Procedure"* is that procedure most closely related to the principal diagnosis (there may be more than one principal procedure). These titles are used in calculating utilization rates, and can be used to retrieve charts for retrospective quality studies, and so on. The data set required for the DRG-Department of Health study uses *"Principal Diagnosis"* to indicate the condition which required admission, but adds another term, *"Major Diagnosis,"* that diagnosis requiring the greatest consumption of hospital resources, including not only utilization, but also costs. If the DRG-prospective reimbursement system is extended to all New Jersey hospitals, this distinction will be important. A mislabeled "major diagnosis"

can put the care in the wrong DRG, and affect the hospital's reimbursement.

To add to the problems, an updated coding system, ICD-9-CM (International Classification of Disease, 9th Version, with Clinical Modifications for the US and Canada), is being brought on line now, and should be in use as of July 1. This will give the medical records people a few more gray hairs.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Office of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ALLERGY—Yalamanchi K. Rao, M.D. 354 Henry Street. Apt. #2, Brooklyn, NY 11201. Nagpur Medical College (India) 1968. Board eligible. Group, partnership, solo, institutional, research. Available July 1979.

Richard W. Huss, M.D. 555 Graham Road, Fort Sam Houston, TX 78234. CMDNJ 1971. Subspecialty, internal medicine. Board certified (IM). Group or solo. Available September 1979.

Andrew W. Green, M.D., 1 Raintree Island, Apt. 6, Tonawanda, NY 14150. St. Louis University 1974. Subspecialty, internal medicine. Board certified (IM). Solo or group. Available July 1979.

ANESTHESIOLOGY—Jitendra G. Patel, M.D., 133-52 Avery Avenue, Flushing, New York 11355. Jamnagar (India) 1972. Board eligible. Any type practice. Available July 1979.

Rolycito A. Chan, M.D., 2121 S. Wolf Road, Hillside, IL 60162. Cebu (Philippines) 1974. Board eligible. Group, partnership, solo. Available July 1979.

Ajit V. Pai, M.D., 8136 Braeburn Terrace, Indianapolis, IN 46219. Topiwala National (India) 1973. Board eligible. Group or partnership. Available September 1979.

Mohammed A. Mohiuddin, M.D., 104 West Franklin Street, Apt. 908, Richmond, VA 23220. Osmania Medical College (India) 1971. Board eligible. Group or partnership. Available July 1979.

Kie C. Ohn, M.D., 520 Second Avenue, Apt. 11-F, New York, NY 10016 Seoul National (Korea) 1971. Subspecialty, family practice. Board eligible. Group, partnership, or solo. Available July 1979.

Vinod Malhotra, M.D., 40 Prospect Avenue, Apt. 63, Hackensack 07601. University of Delhi (India) 1972. Board eligible. Group, partnership, or solo. Available July 1979.

Hyun Hwang, M.D., 435 East 70th Street, Apt 7-F, New York, NY 10021. Yonsei University (Korea) 1964. Board eligible. Group, partnership, solo, or industrial. Available July 1979.

Subbarao Narepalem, M.D., 2947 Northview Drive, Youngstown, OH 44504. Andhra Medical College (India). Board eligible. Group, partnership, fee for service. Available July 1979.

Subba Rao Chennupati, M.D., 407 Lindsay Court #12, Louisville, KY 40206. Rangaraya (India) 1970. Board eligible. Group, partnership, solo. Available July 1979.

CARDIOLOGY—Masoud Javed, M.D., Deborah Heart and Lung Center, Browns Mills 08015. Liaquat Medical College (Pakistan). Subspeciality, internal medicine. Any type practice. Available July 1979.

CARDIOVASCULAR DISEASES—Il Young Kim, M.D., 7044 Glen Meadow Lane, Cincinnati, OH 45237. Seoul National University (Korea) 1971. Subspecialty, internal medicine. Board certified (IM). Group, partnership, institutional. Available July 1979.

Stephen M. Barolsky, M.D., 6347 Caton Street, Pittsburgh, PA 15217. SUNY—Downstate 1973. Subspecialty, internal medicine. Board certified (IM). Group, partnership, institutional, research. Available July 1979.

Rajinder K. Shalla, M.D., 353 East 17th Street, Apt. 8-F, New York, NY 10003. All-India Institute (India) 1968. Subspecialty, internal medicine. Board eligible (IM). Group, institutional. Available July 1979.

Vrajesh M. Parikh, M.D., 2520 South King Drive, Chicago, IL 60616. B.J. Medical College (India) 1970. Subspecialty, internal medicine. Board eligible (IM). Group, partnership, research. Available July 1979.

Shashibalao P. Sehgal, M.D., Rockefeller University, 1230 York Avenue, New York, NY 10021. Seth G.S. Medical College (India) 1973. Subspecialty, internal medicine. Board certified (IM). Group, partnership, public health, industrial, institutional. Available July 1979.

DERMATOLOGY—Fred V. Kemp, M.D., 114 East Bailey Road, Naperville, IL 60540. St. Louis University 1973. Group, partnership, or solo. Available July 1980.

EMERGENCY MEDICINE—Nicanor C. Lopez, M.D., 1432 Arch Street, Apt. 8202, Norristown, PA 19401. Far Eastern University (Philippines) 1967. Subspecialty, general practice. Group, partnership, emergency room. Available July 1979.

ENDOCRINOLOGY—George Tun-I Yang, M.D., 1764 Bising Avenue, #2, Cincinnati, OH 45239. Taipei Medical (Taiwan) 1972. Subspecialty, internal medicine. Board certified (IM). Group, partnership, solo. Available July 1979.

FAMILY PRACTICE—Timothy B. Eckel, M.D., 1410 Bloom Road, Danville, PA 17821. Temple 1976. Board eligible. Group or partnership. Available July 1979.

GASTROENTEROLOGY—Alan M. Nelson, M.D., 41B Monroe Street, Milford, CT 06460. Georgetown 1974. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

Robert S. Goldblatt, M.D., 150 Division Street, Derby, CT 06418. George Washington University 1974. Subspecialty, internal medicine. Board eligible (IM). Group or partnership. Available July 1979.

Jon David Green, M.D., 509 Fairview Avenue, Apt. C-1, Orange 07050. CMDNJ 1974. Subspecialty, internal medicine. Board certified (IM). Group, partnership, solo. Available July 1979.

Hua-Lung Lin, M.D., 911 West Apollo Avenue, Tempe, AZ 85283. National Taiwan University 1972. Subspecialty, internal medicine. Board eligible (IM). Group, partnership, solo. Available July 1979.

Myung Woong Kim, M.D., 6582 Poplar Woods, Circle S., Germantown, TN 38138. Yonsei University (Korea) 1968. Subspecialty, gastroenterology. Board certified (IM). Group, partnership, solo. Available July 1979.

Vithal Kusuma, M.D., 1227 S. Harlem Street, Berwyn, IL 60402. Osmania (India) 1969. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

Ira M. Litzenblatt, M.D., 4B Staunton Court Road, Farmington, CT 06032. Michigan 1975. Subspecialty, internal medicine. Board certified (IM). Group, partnership, associate. Available July 1980.

GENERAL PRACTICE—Dharamsi D. Shah, M.D., 22-4B Passaic Avenue, Nutley 07110. Grant Medical College (India) 1974. Subspecialty, psychiatry. Board eligible (psych.). Group, partnership, solo. Available July 1979.

James W. Warren, M.D., Ainsworth Health Clinic, Fort Hamilton, Brooklyn, NY 11252. Meharry 1973. Group, partnership, rural sponsorship, emergency room. Available.

HEMATOLOGY—Ming-Swang Lok, M.D., 163 Oxford Avenue, Buffalo, NY 14209. National Defense Medical Center (Taiwan) 1970. Subspecialty, internal medicine. Board certified (IM). Research or institutional. Available July 1979.

Subhash C. Proothi, M.D., 86-D Village Green North, East Providence, RI 02915. Rohtak Medical College (India) 1970. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

INFECTIOUS DISEASES—James E. Milder, M.D., 870 Johnsdale Drive, Lexington, KY 40505. Temple 1974. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

INTERNAL MEDICINE—Franciska Katona, M.D., Village Lane Apts., Oak-2, Abington, PA 19001. Temple 1976. Board eligible. Industrial, occupational, research, clinic, preventive medicine. Available July 1979.

Tsue-Pin Lin, M.D., 1945 Corlies Avenue, Neptune 07753. Kaohsiung (Taiwan) 1971. Subspecialty, general practice. Board eligible. Group, partnership, or solo. Available July 1979.

B. Madhusudhan Reddy, M.D., 69 Gold Street, Apt. 12-C, New York, NY 10038. Osmania Medical College (India) 1974. Board eligible. Group, partnership, institutional. Availavle July 1979.

Indu B. Patel, M.D., 9668 Dundawan Road, Baltimore, MD 21236. B.J. Medical College (India) 1971. Board eligible. Group, partnership, institutional. Available July 1979.

Rangachari Srinivasan, M.D., 1325 Pennsylvania Avenue, Apt. 15-D, Brooklyn, NY 11239. Stanley Medical College (India) 1970. Subspecialty, neoplastic diseases. Board certified. Group, partnership, solo. Available July 1979.

Mohamed Ziauddin, M.D., VA Center, Martinsburg, WV 25401. Madurai Medical College (India) 1969. Board eligible. Group, partnership. Available July 1979.

Robert I. Chapnick, M.D., 98-01 67th Avenue, Apt. 12-U, Forest Hills, NY 11374. SUNY—Downstate 1976. Subspecialty, emergency medicine. Board eligible. Group or partnership. Available August 1979.

Bui Ngoc Diep, M.D., 417 NE 13th Street, Oklahoma City, OK 73104. Faculty of Medicine (Vietnam) 1971. Subspecialty, neoplastic diseases. Board eligible. Group, partnership, institutional. Available July 1979.

Imdad H. Butt, M.D., Apt. 5B-1 Franklyn Manor 114 Franklyn Street, Morristown 07960. King Edward (Pakistan) 1970. Subspecialty, general practice. Board eligible (pediatrics). Partnership or solo. Available July 1979.

John S. Smiarowski, M.D., U.S. Naval Hospital, 7500 East Carson Street, Long Beach, CA 90822. Yale 1971. Subspecialty, gastroenterology. Board certified. Group, partnership, solo. Available August 1979.

Fred H.Miller, M.D., 378 Valley Park South, Bethlehem, PA 18018. Jefferson 1975. Board eligible. Group, partnership, solo. Available October 1979.

Rachakonda D. Prabhu, M.D., 80-15 41st Avenue, Apt. 4C8, Elmhurst, NY 11373. R. G. Kar Medical College (India) 1971. Subspecialty, pulmonary diseases. Board certified. Group, partnership, solo, or institutional. Available July 1979.

Samala R. Swamy, M.D., 355 Bard Avenue, Apt. 32, Staten Island, NY 10310. Kakatiya Medical College (India) 1972. Subspecialty, cardiovascular diseases. Board certified. Group, solo, or institutional. Available July 1979.

Jay H. Alexander, M.D., 30 Waterside Plaza, Apt. 9-E, New York, NY 10010. Loyola 1976. Subspecialty, emergency medicine. Board eligible. Industrial, emergency room, school health, institutional. Available July 1979.

Joel S. Steinberg, M.D., 6641 Oakland Street, Philadelphia, PA 19149. Temple 1976. Subspecialty, family practice. Board eligible. Group or partnership. Available July 1979.

N. Satish Kamath, M.D., 401 Tobin Drive, #310, Inkster, MI 48141. Karnatak Medical College (India) 1972. Board eligible. Group, partnership, solo. Available August 1979.

Sambaiah Kankanala, M.D., 122 Ashland Place, Apt. 11-N, Brooklyn, New York 11201. Osmania Medical School (India) 1972. Subspecialty, pulmonary diseases. Board certified. Group or solo. Available July 1979.

Nadakavil M. Koshy, M.D., 8 Slate Creek Drive, Apt. 9, Cheektowaga, NY 14227. Kottayam Medical College (India) 1971. Subspecialty, cardiovascular diseases. Board certified. Group, partnership, solo, or institutional. Available July 1979.

Chakumkal Cyriac,M.D., 3001 South Hanover Street, Baltimore, MD 21230. T.D. Medical College (India) 1973. Subspecialty, general practice. Board eligible. Group, partnership, solo. Available August 1979.

George T. Yang, M.D., 1764 Bising Avenue, Apt. 2, Cincinnati, OH 45239. Taipei (Taiwan) 1972. Subspecialty, endocrinology. Board certified. Group, partnership, solo, or emergency room. Available July 1979.

Pervaiz Rahman, M.D., 1650 Grand Concourse, Bronx, NY 10457. Dow Medical College (Pakistan) 1972. Subspecialty, gastroenterology. Board eligible. Group, partnership, institutional. Available July 1979.

Jambur E. Chandrashekar, M.D., 155 Forest Road, #39, St. John's A1A 1E7, Newfoundland, Canada. Mysore Medical College (India) 1970. Board eligible. Group, partnership, solo, or institutional. Available July 1979.

Jerome W. Takiff, M.D., 2917 E. Turquoise, Phoenix, Arizona 85028. Cornell 1973. Board certified. Group. Available.

Alan J. Sten, M.D., 105 West 13th St., Apt. 15-C, New York, NY 10011. New York Medical (1972). Subspecialty, infectious diseases. Board certified. Partnership or group. Available July 1979.

Pervaiz Rahman, M.D., 240 Yonkers Avenue, Apt. 1-C, Yonkers, NY 10701. Dow (Pakistan) 1972. Subspecialty, gastroenterology. Board eligible. Group, partnership, solo, or institutional. Available July 1979.

Aziz H. Junagadhwalla, M.D., 9 Nixon Court, Apt. 3-J, Brooklyn, NY 11223. Grant Medical College. Subspecialty, pulmonary medicine. Group or partnership. Available July 1979.

Soma N. Pulipati, M.D., 725 East Main Street, Kings Park, NY 11754. Osmania (India) 1971. Subspecialty, cardiology. Board certified. Solo, group, partnership (in that order). Available July 1979.

Richard E. Handelsman, D.O., 40 Prospect Avenue, Apt. 3-M, Bldg. 1, Norwalk, CT 06850. College of Osteopathic Medicine & Surgery 1976. Group or partnership. Available August 1980.

Charles F. McNally, M.D., Box 1774, 61 Moulton Road, Duxbury, Massachusetts 02332. New York Medical College 1968. Board certified. Group or partnership. Available July 1979.

Carol D. Silver, M.D., 1619 Third Avenue, Apt. 4-G, New York, NY 10028. Duke 1970. Subspecialty, medical oncology. Board certified. Solo, group, or partnership. Available August 1979.

NEOPLASTIC DISEASES—Victor D. Ribeiro, M.D., 82 Beacon Hill, Apt. 1B3, Dobbs Ferry, NY 10522. Goa Medical College (India) 1970. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

Gregory P. Burke, M.D., 45 Viscount Road,Longmeadow, MA 01106. SUNY—Downstate 1972. Subspecialty, internal medicine. Board Certified (IM). Group or academic. Available.

NEUROLOGY—Hungi Papanna Eswara, M.D., 843 School Lane House, 5450 Wissahickon Avenue, Philadelphia, PA 19155. Government Med. College (S. India) 1971. Board eligible. Solo, group, partnership. Available July 1979.

OBSTETRICS/GYNECOLOGY—Devendra M. Bhardwaj, M.D., 242 Gurdon Street, Bridgeport, CT 06606. S.M.S. Medical College (India). Board eligible. Group, partnership, solo, HMO. Available July 1979.

Kenneth Garrett, M.D., 31-A1 Lakeshore Drive, Farmington, CT 06032. NYU 1975. Board eligible. Group, partnership. Available August 1979.

Charles Ackerman, M.D., 353 East 17th Street, New York, NY 10003. Tel Aviv (Israel) 1975. Board eligible. Group or partnership. Available September 1979.

Kok C. Chang, M.D., 411 West 114th Street, Apt. 4-B, New York, NY 10025. National Taiwan University 1973. Board eligible. Group, partnership, solo. Available August 1979.

Sang Bak Yoo, M.D., 10856 Nottingham, Detroit, MI 48224. Kyung-Pook University (Korea) 1969. Board eligible. Group, partnership, or solo. Available July 1979.

Steven B Wirts, M.D., 340 Nina Street, New Windsor, NY 12550. Jefferson 1974. Board eligible. Group or partnership. Available July 1979.

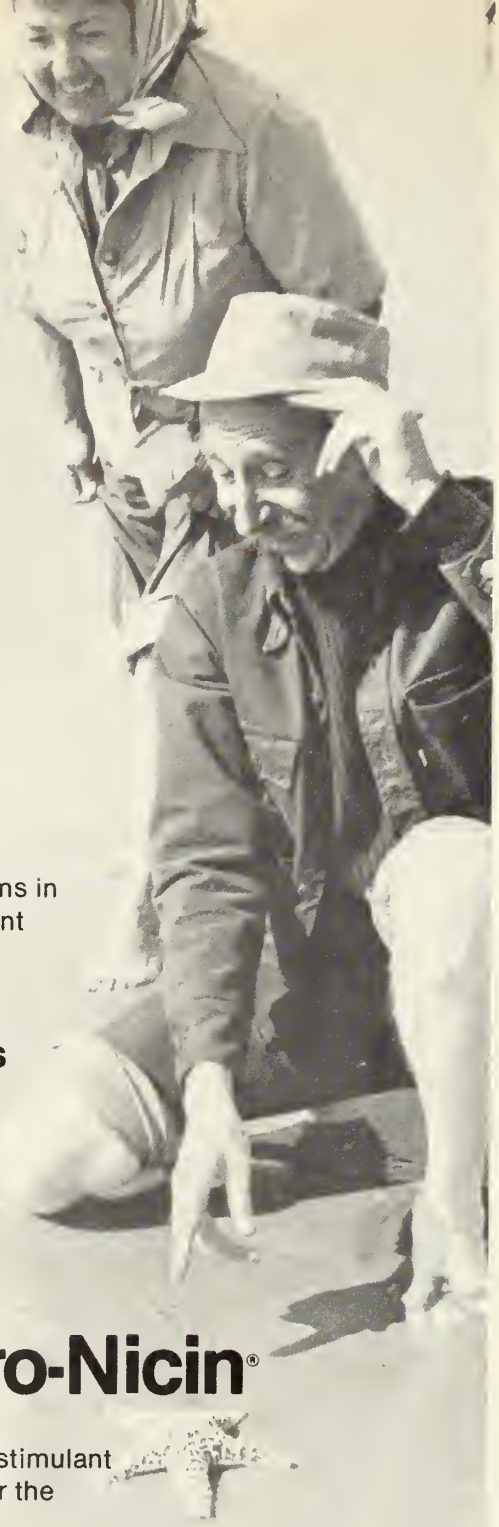
OCCUPATIONAL MEDICINE—Arthur E. Mittnacht, M.D., 299 Edgerstoune Road, Princeton 08540. Columbia 1945. Subspecialty, orthopedic surgery. Board certified (orthopedic surg.) Industrial, school health, emergency room. Available May 1979.

OPHTHALMOLOGY—Jonathan F. Wise, M.D., 1175 York Avenue, Apt. 4-H, New York, NY 10021. SUNY—Buffalo 1973. Subspecialty, pediatrics. Board eligible. Group or partnership. Available June 1979.

Joshua Frankel, M.D., 161 North Sycamore Avenue, Los Angeles, CA 90036. Maryland 1971. Board certified. Partnership, solo, institutional. Available June 1979.

Alan 1. Mandelberg, M.D., 284 Wintergreen Way, Rochester, NY 14618. SUNY—Buffalo 1972. Board certified. Group, partnership, or solo. Available July 1979.

Raphael J. Sapeika, M.D., 7 Windsor Road, Great Neck, NY 11021. Cape Town (South Africa) 1970. Board eligible. Group or partnership. Available July 1979.



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ORTHOPEDICS—Upendra Kumar Sinha, M.D., 86-16 60th Avenue, Apt. 2-E, Rego Park, NY 11373. Prince of Wales (India) 1968. Board eligible. Group, partnership, solo. Available July 1979.

OTOLARYNGOLOGY—Vijay Chandra Singh Bais, M.D., 2174 Green Spring Drive, Kettering, OH 45440. Mahatma Gandhi (India) 1961. Board certified. Solo, partnership, group. Available.

OTORHINOLARYNGOLOGY—Jang-Huei Jang, M.D., 7563 Drumheller Drive, Richmond, Virginia 23225. Chung-Shan (Taiwan) 1967. Board eligible. Group or solo. Available July 1979.

Gregory J. Milmoe, M.D., 259 Canner Street, New Haven, CT 06511. Chicago 1973. Board eligible. Group, research, partnership. Available August 1979.

PATHOLOGY—Mansoora R. Chaudry, M.D., 41-04 Goldblatt Terrace, Fair Lawn 07410. Fatima Jinnah Medical College (Lahore, India). Board certified. Any type practice. Available.

Dhanasaran Mongkolsmai, M.D., 900 West Spencer Street, Apt. 302, Philadelphia, PA 19141. Ramathibodi (Thailand) 1971. Special interest, clinical pathology. Board certified. Group, partnership, institutional. Available September 1979.

Ahm Abdul Mannan, M.D., 215 East 77th Street, Apt. 3-C, New York, NY 10021 Dacca (Pakistan) 1961. Special interest, clinical pathology. Board eligible. Group, research, institutional. Available July 1979.

Luis R. Molinas, M.D., 3041 West Chester Pike, Apt. G-7, Broomall, PA 19008. National University (Paraguay) 1972. Board eligible. Partnership, group, institutional. Available July 1979.

Ruly Yoediono, M.D., 10304 East Malcolm Circle, Cockeysville, MD 21030. Padjadjaran (Bandung) 1971. Special interest, clinical pathology. Board eligible. Group, partnership. Available September 1979.

Mehboob M. Fatteh, M.D., 3405 Crane Ferry Road, Augusta, GA 30907. H.H.L. Medical College (India) 1968. Board eligible. Group, partnership, solo, institutional. Available March 1979.

Frank R. Rudy, M.D., 7614 Woodcrest Avenue, Philadelphia, PA 19151. Pittsburgh 1974. Board eligible. Institutional, partnership, research. Available July 1979.

PEDIATRICS—Georgino R. Reyes, M.D., 18 Palmetto Circle, Brownsville, TX 78521. Santo Domingo (Dominician Republic) 1960. Board certified. Group, partnership, research. Available July 1979.

Jesse M. Mintz, M.D., 265-24 74th Avenue, Apt. G-1, Glen Oaks, NY 11004. Columbia 1972. Board certified. Institutional, group. Available July 1979.

Ganesh N. Deshpande, M.D., 49 Stoneledge Court, Williamsville, NY 14221. Grant Medical College (India) 1970.

Subspecialty hematology. Board eligible. Institutional, research, group. Available July 1979.

Chalermlarp Mongkolsmai, M.D., 900 West Spencer Street, Apt. 302, Philadelphia, PA 19141. Ramathibodi Medical School (Thailand) 1971. Subspecialty, pediatric cardiology. Board certified. Institutional, research, group. Available September 1979.

Theodore H. Sher, M.D., 170 West End Avenue, New York, NY 10023. SUNY—Downstate 1974. Subspecialty, pediatric allergy. Board eligible. Group, partnership. Available July 1979.

Nam Soo Lee, M.D., Meadowbrook Apt. 44, Blessing Road, Slingerlands, NY 12159. Seoul National University (Korea) 1971. Subspecialty, pediatric cardiology. Board eligible. Solo, group, research. Available July 1979.

Elias G. Gouel, M.D., 6036 East Pratt, Baltimore, MD 21224. Ain Shams University (Egypt) 1971. Board eligible. Group, partnership. Available August 1979.

Ira B. Toback, M.D., 4618 Avenue J. Brooklyn, NY 11234. Louvain (Belgium) 1976. Board eligible. Partnership or group. Available July 1979.

Boo Woong Lee, M.D., 1160 Midland Avenue, Apt. 8-M, Bronxville, NY 10708. Yonsei (Korea) 1973. Board eligible. Group, partnership, solo, institution. Available July 1979.

Roy Gomez, M.D., 300 Lookout Avenue, Apt. D-9, Hackensack 07601. Trivandrum (India) 1971. Board eligible. Group, partnership, institution, solo. Available July 1979.

PHYSICAL MEDICINE/REHABILITATION—Diane C. Griffin, M.D., 29 East 29th Street, New York, NY 10016. University of Rome (Italy) 1974. Subspecialty, general practice Board eligible. Institutional, group, research. Available July 1979.

PSYCHIATRY—Sumanlal J. Kaneria, M.D., P.O. Box 9587, Cheyenne, WY 82001. M.P. Shah Medical School (India) 1971. Board eligible. Academic type practice. Available October 1979.

Balasubramaniam Gulasekaram, M.D., Apt. 1-D, Beechwood Hall, Westchester City Medical Center, Valhalla, NY 10595. Colombo (Sri Lanka) 1970. Board eligible. Research, institutional, group. Available September 1979.

Herman Berliss, M.D., 110 Boteler Street, Apt. 703, Ottawa, Ontario K1N 8W8, Canada. Case Western Reserve 1974. Board eligible. Adult outpatient. Available July 1979.

PULMONARY DISEASES—Joseph I. Matthews, M.D., OSC Box 553, Walter Reed Hospital, Washington, DC 20012. Creighton 1969. Subspecialty, internal medicine. Board certified (IM). Group, institutional, partnership. Available August 1979.

Richard T. Bell, M.D., 1740 Aidenn-Lair Road, Dresher, PA 19025. Jefferson 1972. Subspecialty, internal medicine. Board certified (IM). Group, solo. Available July 1979.



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Harjinder S. Gogia, M.D., 6201 North 10th Street, Apt. 435, Philadelphia, PA 19141. Punjabi University (India) 1973. Subspecialty, internal medicine. Board certified (IM). Group, institutional. Available July 1979.

RADIOLOGY—Seetharam A. Mattupalli, M.D., 7083 Middle Valley Walk, Apt. B, St. Louis, MO 63123. Guntur (India) 1971. Subspecialty, diagnostic radiology. Board eligible. Partnership, research, group, administrative, institutional. Available July 1979

Henry Mang, M.D., 8 Predmore Avenue, Colonia 07067. Sydney (Australia) 1965. Subspecialty, nuclear medicine. Board certified. Any type practice. Available.

Patrick F. Zazzaro, M.D., 6839 Shawnee Road, Richmond, VA 23225. CMDNJ-Rutgers 1975. Particular interest—diagnostic radiology. Board eligible. Group, partnership. Available July 1979.

RHEUMATOLOGY—Carlos Ganuza, M.D., 2D Beaver Court, Wilkes-Barre, PA 18702. University of El Salvador 1971. Subspecialty, internal medicine. Board eligible (IM). Institutional, group. Available July 1979.

Stuart P. Brogadir, M.D., 6357 Lancaster Avenue, Philadelphia, PA 19151. Cornell 1975. Subspecialty, internal medicine. Board eligible (IM). Research, group. Available July 1979.

SURGERY, CARDIOVASCULAR—Michael L. Weinberger, M.D., 5902 Weatherford Road, Fayetteville, NC 28303. SUNY—Downstate 1971. Subspecialty, thoracic surgery. Board eligible (general surgery). Group or partnership. Available June 1979.

SURGERY, COLON/RECTAL—Antoine Sara, M.D., 2745 Birchcrest Drive, SE, Apt. 209, Grand Rapids, MI 49506. Damascus (Syria) 1971. Group, partnership, solo. Available August 1979.

SURGERY, GENERAL—Clementino O. Purisima, M.D., 105 Chadwick Road, Teaneck 07666. Santo Tomas (Philippines) 1969. Board eligible. Group partnership, solo. Available July 1979.

John L. Deboer, M.D., 1242B Cedar Street, Fort Dix 08640. Vermont 1971. Board certified. Group or partnership. Available June 1979.

Joel A. Yellin, M.D., 215-16 15th Road, Bayside, New York 11360. Rochester 1973. Board eligible. Partnership, solo, group. Available July 1979.

Glenn A. Pasternack, M.D., 7660 Phoenix Drive, Apt. 1559, Houston, TX 77030. SUNY—Downstate 1974. Board eligible. Partnership, group. Available July 1979.

Marc A. Grossman, M.D., 21140 18th Avenue, Bayside, NY 11360. Einstein 1975. Board eligible. Partnership or group. Available July 1979.

Dilipkumar H. Kakaiya, M.D., Lafayette Towers, Apt. 709, 2040 Lehigh Street, Easton, PA 18042. Grant Medical College (India) 1971. Board eligible. Solo, partnership, group. Available July 1979.

Bassam T. Kashlan, M.D., 65 Cedar Avenue, Apt. D-16, Long Branch 07740. Damascus (Syria) 1972. Board eligible. Partnership, institutional, group. Available July 1979.

Frederick J. Heinle, Jr., M.D., 328 S. Linden Avenue, Pittsburgh, PA 15208. Board eligible. Partnership, group, solo. Available July 1979.

Stephen F. Winer, M.D., Box 633, Wiesbaden, West Germany, APO New York 09220. Wayne 1967. Board certified. Partnership, group. Available January 1980.

Meherwan J. Mistry, M.D., 524 Morris Avenue, Apt. 3-E, Elizabeth 07208. Grant Medical College (India) 1972. Board eligible. Partnership, group, solo. Available July 1979.

Ivan A. Shulman, M.D., 785 Burnett Avenue, Apt. 7, San Francisco, CA 94131. Pittsburgh 1972. Board eligible. Group, partnership. Available July 1979.

Vithal D. Kardani, M.D., 133-52 Avery Avenue, Flushing, NY 11355. Jamnagar (India) 1972. House physician or residency. Available.

Vincent Larkin, Jr., M.D., 7021 Colonial Road, Brooklyn, NY 11209. SUNY-Downstate 1974. Subspecialty, vascular surgery. Board eligible. Group or partnership. Available July 1979.

Geoffrey Simon, M.D., RFD 2, Box 155, Harrison, Maine 04040. Boston 1968. Board certified. Group, partnership, solo. Available.

K. Venkataraman, M.D., 380 North Broadway, Apt. C-14, Yonkers, NY 10701. Madras (India) 1966. Board eligible. Solo or group. Available July 1979.

SURGERY, ORTHOPEDIC—Stefan Glowacki, M.D., 2130 Williamsbridge Road, Bronx, NY 10461. Warsaw (Poland) 1962. Subspecialty, orthopedic surgery. Board eligible (orth. surg.) Solo, partnership, research. Available July 1979.

Ronald J. Leavitt, M.D., 717 Willow Way, Narberth, PA 19072. Columbia 1971. Subspecialty, hand surgery. Board certified. Partnership, group. Available July 1979.

Samir R. Dawoud, M.D., 1120B Easton Avenue, Bldg. 18, Somerset 08873. Kasr El Aini (Egypt) 1960. Board eligible. Partnership, group. Available July 1979.

SURGERY, UROLOGICAL—Robert J. Claire, M.D, 111 Annadale Road, Staten Island, NY 10312. Guadalajara (Mexico) 1974. Special interest in family counseling. Any type practice. Available July 1980.

Natvarlal P. Patel, M.D., 404 Sixth Stteet, Apt. 5D, Coraville, IA 52241. B. J. Medical School (India) 1970. Subspecialty, general surgery. Board eligible. Solo, partnership, group, research. Available July 1979.

Daniel H. Piazza, M.D., 741 Peninsula Court, Ann Arbor, MI 48105. Illinois 1972. Board eligible. Group or partnership. Available July 1979.

Sheldon Muhlbauer, M.D., 3450-23 Wayne Avenue, Bronx, NY 10467. Einstein 1973. Board eligible. Partnership, group. Available July 1979.

Shree Subhash, M.D., 8 Sugarloaf Court, Baltimore, MD 21209. Prince of Wales Medical College (India) 1967. Partnership, group, solo. Available September 1979.

UROLOGY—H. Barry Opell, M.D., 1275 East 51st Street, Brooklyn, NY 11234. Lausanne (Switzerland) 1971. Board certified. Solo, partnership, group. Available.

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Position Papers—Psychiatric Association

The following position papers—"Statement on Functions of a Psychiatrist" and "Statement on Active Treatment"—were adopted by the American and New Jersey Psychiatric Associations.

STATEMENT ON FUNCTIONS OF A PSYCHIATRIST

The American Psychiatric Association, on behalf of all persons in need of medical services, is dedicated to the principle that, under any comprehensive health insurance program, there must be coverage for both mental and physical illness sufficient to meet the medical needs of each patient. Medical need is highly individual, and is defined operationally in each case by the evaluation, diagnosis, and recommendation evolved in the interaction between the treating physician and his patient. Excessive administrative or fiscal intrusion into this process is antithetical to good medical practice. The following statements on the functions of psychiatry and on insurance coverage for mental illness call attention to the fact that psychiatrists are the only persons qualified by education, training, and experience to evaluate and treat the total biological, psychological, and social entity called the person. Furthermore, the physician is and long has been designated by the society as the only person responsible for the overall treatment of the patient.

A. Psychiatrists are medical specialists who:

(1) are Board Certified by the American Board of Psychiatry and Neurology; or

(2) successfully have completed a minimum of three years of approved graduate medical training in psychiatry.

B. The psychiatrist is the physician member of the mental health team, and as such, his or her function is different from the activities of other mental health professionals.

The psychiatrist has, but is not limited to, the following characteristics:

(1) General medical training prior to undertaking specialized work in the field of psychiatry.

(2) An understanding that mental illness, like physical disease, has multiple causes, involving biological, social, and psychological factors. In no other discipline is this multifactorial approach to diagnosis and treatment so basic.

(3) The qualifications to integrate physical, emotional, personality, and other psychological factors in the differential diagnosis of illness.

(4) Neurological training which is important in differentiating organic from psychological and emotional etiological factors.

(5) Specialized training in psychopharmacology, including an understanding of the idiosyncratic physical and psychological reactions of patients to drugs.

(6) Unique skills to supervise patient management because of his or her combined medical and psychological training.

(7) Special training to handle emergencies arising in the course of the treatment of mental illness.

(8) The qualifications to treat the most difficult cases as well as milder distresses.

(9) The unique qualification to deal with psychotic illnesses, because of his or her specialized training, the ability to hospitalize patients and prescribe medications, and the ability to correlate severe psychological dysfunction with physical and biochemical data.

(10) The best liaison with the medical community for cross referral, consultation, teaching, and treatment planning.

(11) The understanding of the effects of physical illness and its emotional complications.

(12) The capacity to prescribe other therapies as supplemental to his or her own treatment program, or as an alternative to it.

(13) The ability to carry out ongoing evaluation and modification of the patient's total treatment.

(14) The unique qualification of being the only mental health professional designated by society and tradition, and uniquely prepared by training and experience, to accept medical-legal responsibility.

C. Under comprehensive health insurance, funds should be provided for whatever form of treatment is indicated to meet the medical needs of each patient.

STATEMENT ON ACTIVE TREATMENT

Psychiatric treatment is active treatment, a planned effort on behalf of persons defined either by themselves or by their community as mentally ill or emotionally disturbed and in need of treatment. The person directing it must be qualified by special education and training to evaluate and understand the totality of the biological, psychological, and social factors which play a part in such an illness. Treatment is provided through medical procedures designed to benefit the ill person.

General Principles

1. Treatment may begin prior to the establishment of a final diagnosis. The process of evaluation is an act of treatment.

2. The standards used by a community to judge behavior may not always be in agreement with the standards used by a psychiatrist to judge behavior. For example, some people judged by community standards to be "bad" rather than ill may suffer from a diagnosable mental illness. On the other hand, some persons whose behavior is identified as aberrant by a given community may be perceived by the psychiatrist as following an alternative life style and not as suffering from an illness.

3. As in physical illness, an individual's subjective distress may in itself be sufficient justification for treatment.

4. Psychiatric disorders result from the complex interaction of physical, psychological, and social factors and treatment may be directed toward any or all three areas.

5. Treatment may include measures to maintain current functioning and prevent further deterioration as well as measures designed to improve or eliminate dysfunction.

6. A variety of professional disciplines may be involved in a treatment program. The extent and kind of participation of any practitioner in a specific treatment program should be determined by the person primarily responsible for providing treatment. Professional qualifications and ethics of the various disciplines are defined by each professional group within society's sanctions. No practitioners should be required to participate in a manner contrary to the ethic of their discipline.

7. A formal or informal treatment plan is an integral part of treatment. The plan should include the goals of treatment, problems which may be anticipated, and should be revised when appropriate and indicated. Psychiatric treatment should be based on principles which can be explained and communicated during review by one's peers.

8. Providing a humane environment for the care of persons in need is not equivalent to providing treatment. However, when the environment is organized carefully to respond in a therapeutic manner to patients' needs and behavior, and is staffed and supervised by qualified members of appropriate professional disciplines, it is a form of treat-

ment. Treatment of that kind usually is referred to as a therapeutic environment or milieu therapy.

9. Psychiatric treatment is the sum of the activities of a psychiatrically qualified physician in meeting the therapeutic needs of a patient or family. This may include the supervision of others who are providing treatment and for whose activities the psychiatrist accepts professional and legal responsibility.

Correction

The Journal calls attention to an error in the text and in placement of two illustrations in the article, "Ultrasound Evaluation of a Spermatic Cord Myxoliposarcoma"—Gould; Klein; Patel, which appears in the March 1979 issue of *JMSNJ*, pages 203-205. On page 203, righthand column, fifth line from the bottom, the sentence starting in the middle of the line should read: "A high gain view was obtained" On page 204 illustrations 2 and 3 should be transposed so as to appear over their proper legends.

Area Map—MSNJ Headquarters



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CME CALENDAR

This listing is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, the Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the College of Medicine and Dentistry of New Jersey. For information on accreditation, please contact the sponsorings organization(s), indicated by italics—last line of each item.

MEDICINE (includes Family, Internal, and General Medicine and Dermatology)

June

1 **Adrenal Diseases**
8:30-9:30 a.m.—United Hospitals of Newark
(United Hospitals of Newark and AMNJ)

5 **Weekly Tumor Conferences**
12 11 a.m.-12 noon—Morristown
19 Memorial Hospital
26 *(Morristown Memorial Hospital and AMNJ)*

6 **Advances in Medicine**
13 9:30 a.m.-11 a.m.—Bergen Pines County
20 Hospital, Paramus
(Bergen Pines County Hospital and AMNJ)

6 **Hypoglycemia**
13 **Diseases of the Esophagus**
9:30-11:30 a.m.—Dover General Hospital
(Riverside, Dover General, St. Clare's Hospitals and AMNJ)

6 **Endocrine Conferences**
3:30-5 p.m.—Rotates between Newark Beth Israel Medical Center, Martland Hospital, Newark, and VA Hospital, East Orange
(AMNJ Endocrinology Section)

6 **Clinical Shock**
11:30 a.m.—Rahway Hospital
(Rahway Hospital and AMNJ)

6 **Echocardiography**
13 **Psychotropic Medication**
10:30 a.m.-12 noon—St. Mary's Hospital, Passaic
(St. Mary's Hospital and AMNJ)

6 **Diabetic Coma and Pre and Postoperative Diabetic Care**
13 **Hypertension**
20 **Medical Lecture Series**
27 1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

7- **Lectures**
10 Southampton Princess Hotel, Bermuda
(NJ Dermatological Society and AMNJ)

7 **Anemia**
12 **Anemia in Pregnancy**
14 **Leukemia**
21 **Blood Disease Tests**
28 **Drug Interactions**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

7 **Cardiology Conferences**
14 2:15-4:15 p.m.—Deborah Heart and
21 Lung Center, Browns Mills
28 *(Deborah Heart and Lung Center and AMNJ)*

8- **Otolaryngology for Primary**
9 **Physicians**
Playboy Club, McAfee
(NJ Medical School, United Hospitals Medical Center and AAFP)

12 **Lower Back Pain**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

14 **Case Presentations and Guest Speakers**
7:30-9:30 p.m.—Location to be announced
(NJ Institute of Ultrasound in Medicine and AMNJ)

19 **Peripheral Vascular Disease**
11:30 a.m.-12:30 p.m.—St. Mary's Hospital, Orange
(St. Mary's Hospital and AMNJ)

19 **Cardiac Arrhythmias**
8:30 p.m.—Omar's Restaurant, Fair Lawn
(Fair Lawn Memorial Hospital and AMNJ)

21 **Regional Chest Conferences**
7:30-9:30 p.m.—Newark Beth Israel Medical Center
(NJ Thoracic Society and AMNJ)

21 **Infectious Diseases**
4-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

26 **Scanning, New Developments**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

27 **Cardiac Grand Rounds**
3-4:30 p.m.—CMDNJ-NJ Medical School
(American Heart Association, NJ Affiliate and AMNJ)

July

13- **ECG Interpretation and**
15 **Arrhythmia Management**
Playboy Resort, George McAfee
(International Medical Education Corp. and AAFP)

NEUROLOGY/PSYCHIATRY

June

4 **Anorexia Nervosa in an Adult Married Woman**
8-10 p.m.—9 Marquette Road, Upper Montclair
(Essex Psychiatric Seminar and AMNJ)

6 **Depression-Outpatient Treatment**
13 **Psychodynamic Therapy for Outpatients**
1-3 p.m.—Ancora Psychiatric Hospital, Hammonton
(Ancora Psychiatric Hospital and AMNJ)

7 **Improving Psychotherapeutic Techniques**
8-10 p.m.—312 Harding Drive, So. Orange
(Group for Advanced Psychiatric Study and AMNJ)

13 **Unusual Psychiatric Disorders and Atypical Psychoses**
1:15-2:45 p.m.—Marlboro Psychiatric Hospital
(Marlboro Psychiatric Hospital and AMNJ)

13 **Psychiatry in the United States**
1:30-3 p.m—NJ Medical School, Newark
(CMDNJ and AMNJ)

14 **Lecture Series**
8:30-10:30 p.m.—Hackensack Hospital
(NJ Psychoanalytic Society and AMNJ)

19 **Clinical Shock**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

20 **Sexual Medicine**
1:30-3 p.m.—VA Medical Center, Lyons
(VA Medical Center and AMNJ)

OBSTETRICS/GYNECOLOGY

June

2 **Controversies in Obstetrics**
8:30 a.m.-1 p.m.—Clara Maass Memorial Hospital, Bellville
(Clara Maass Hospital and AMNJ)

6 **Grand Rounds and Case Presentations**
13 2-4 p.m.—Rotating between Martland
20 Hospital, Newark Beth Israel, St.
27 Michael's, and St. Joseph's Hospitals, Newark, and Jersey City Medical Center
(CMDNJ and AMNJ)

7 **Grand Rounds and Case Presentations**
14 4-5 p.m.—Martland Hospital, Newark
21 *(CMDNJ and AMNJ)*
28

Gynecologic Endoscopy
14 8 a.m.-5 p.m.
15 7:45 a.m.-4:30 p.m.
Memorial General Hospital, Union
(New Jersey Fertility Foundation and AMNJ)

PATHOLOGY/CLINICAL PATHOLOGY

June

6 **Clinical Pathology Grand Rounds**
13 12 noon-1 p.m.—NJ Medical School,
20 Newark
(CMDNJ and AMNJ)

27 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

SURGICAL SPECIALITIES (includes ENT, neurosurgery, ophthalmology, orthopedic, plastic, and vascular surgery)

June

1 **Orthopedics**
8 7:30-9 a.m.—Alexian Brothers Hospital,
22 Elizabeth
(Alexian Brothers Hospital

Second Annual Symposium on Vascular Surgery
4 8:30 a.m.-5 p.m.
5 9 a.m.-12 noon—The Meadowlands Hilton Harmon Cove
(Englewood Hospital Vascular Surgical Service, Vascular Surgical Society of NJ and AMNJ)

7 **Neurosurgical Case Presentations**
14 4-5:30 p.m.—NJ Medical School,
21 Newark
(CMDNJ and AMNJ)

20 **Management of Face Turns with Nystagmus**
7:15-9:30 p.m.—United Hospitals Medical Center, Annex Bldg., Newark
(Associated Eye Residencies of NJ and AMNJ)

26 **Cervical Spine Injuries**
6-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

MISCELLANEOUS

June

26 **PSRO**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

Letter to the Journal

February 23, 1979

Dear Dr. Krosnick:

I was delighted to receive the copy of *The Journal* of the Medical Society of New Jersey.

I am proud to see the Boehm porcelain Trilafon head on the cover. It brought back many poignant memories. If only Schering knew how much they helped us to meet our payroll in those days—but I know they were happy to do it.

I read the article with interest. What a team the genius of Mr. Boehm and Dr. Franz Goll would have made.

Again, my thanks and gratitude for your thoughtfulness in sending this to my attention.

signed (Helen F. Boehm)

OBITUARIES

Dr. Philip S. Avery

Philip Sutton Avery, M.D., a senior member of our Middlesex County component, died on February 16. Born in Iowa in 1898, Dr. Avery earned his medical degree from St. Louis University, class of 1924. His specialty was radiology, which he practiced in the New Brunswick area until retirement in 1975. Dr. Avery was a Fellow of the American College of Radiology, a member of the New Jersey State Radiological Society, and he had served as a counsellor for the Radiological Society of North America, State of New Jersey. He had been affiliated with Middlesex General and St. Peter's Hospitals in New Brunswick and Princeton Hospital. Doctor Avery was a recipient of MSNJ's Golden Merit Award in 1974.

Dr. Siegbert Bornstein

Siegbert Bornstein, M.D., a member of our Passaic County component, died on March 11. Born in Berlin, Germany in 1903, Dr. Bornstein was a graduate of the medical school at the University of Berlin, class of 1928. He emigrated to the United States and became a citizen in 1943. Dr. Bornstein was board certified in pathology and practiced that specialty in Pompton Lakes. His particular interest was in clinical pathology and pathologic anatomy. He was a Fellow of the American College of Physicians and a consultant at Chilton Hospital in Pompton Plains. Dr. Bornstein served for three years in the medical corps of the AUS during World War II, attaining the rank of major.

Dr. Charles L.S. Brennan

A senior member of our Camden County component, Charles L.S. Brennan, M.D., died on February 21. A native of New Jersey, born in 1898, Dr. Brennan had a brief experience in the United States Army's infantry division in World War I, terminated by the Armistice. Following that he embarked on a medical career, graduating from Jefferson Medical College in 1924. He established a practice in general medicine, later designated as family medicine, in Gloucester. Dr. Brennan was affiliated with Cooper Medical Center in Camden and Underwood Memorial Hospital in Woodbury. He was a recipient of MSNJ's Golden Merit Award in 1974.

Dr. Aram A. Captanian

Word just has been received of the death on August 11, 1978, of Aram A. Captanian, M.D., a member of our Monmouth County component. Born in Armenia in 1912, Dr. Captanian emigrated to the United States and earned his medical degree at Columbia University's College of Physicians and Surgeons, graduating in 1936. He established a practice in general surgery in Matawan in 1938 and prior to his retirement in 1975 had been affiliated with Monmouth Medical Center, Riverview Hospital in Red Bank, and Bayshore Community Hospital in Holmdel. Dr. Captanian served in the medical department of the United States Army for four years during World War II.

Dr. Harry Feinberg

On March 1, Harry David Feinberg, M.D., a member of our Monmouth County Component, died at the Eatontown Convalescent Center. Born in Lithuania, Dr. Feinberg emigrated to the United States as a young child, and after graduating from Rutgers University had the unique experience of working for several years with Thomas A. Edison at the inventor's laboratory in West Orange. He then went on to pursue a career in medicine, earning his degree from New York University, class of 1928. He established a practice in family medicine in Long Branch and was affiliated with Monmouth Medical Center there. Dr. Feinberg served as school doctor in Long Branch for 20 years. During World War II he served in the medical department of the AUS, attaining the rank of major. Dr. Feinberg received MSNJ's Golden Merit Award in 1978, signifying 50 years of service as a physician. He was 79 years old at the time of his death.

Dr. Louis Grunt

Louis Grunt, M.D., an emeritus member of our Essex County component, died in Philadelphia on March 14. A native of New Jersey, Dr. Grunt earned his medical degree from Jefferson Medical College, graduating in 1934. Board certified in internal medicine, he practiced that specialty in Newark, later moving his office to East Orange. Dr. Grunt was a Fellow of the American Diabetes Association, and had been affiliated with East Orange General and Newark City Hospitals. During World War II he served in the medical department of the United States Army. Dr. Grunt was 70 years old at the time of his death.

Dr. Charles F. Hadley, Jr.

Word has been received of the sudden death on February 21 of Charles Frazer Hadley, Jr., M.D., a general practitioner in Westmont. A native of New Jersey, Dr. Hadley was graduated from Hahnemann Medical College in 1932. He was affiliated with West Jersey Hospital in Camden. During World War II he served with the United States Coast Guard. Dr. Hadley was 73 years old at the time of his death.

Dr. Thomas F. Magovern

After a prolonged illness, Thomas F. Magovern, M.D., a senior member of our Essex County Component, died on January 31 at Jersey Shore Medical Center. Born in 1902, Dr. Magovern received his medical degree from the University of Maryland, class of 1930. He established a practice in orthopedic surgery in South Orange, specializing in traumatic surgery. Dr. Magovern had hospital staff appointments at St. Mary's Hospital and The Hospital Center at Orange until illness necessitated his retirement in 1975. That same year, in recognition of his service, the board of trustees at the Hospital Center at Orange promoted Dr. Magovern to honorary staff in their orthopedic unit. Dr. Magovern served in the United States Navy during World War II, emerging with the rank of commander.

Dr. A. Alfred Podell

One of Monmouth County's senior members, A. Alfred Podell, M.D., of Red Bank, died on March 7 at Riverview Hospital. Born in New York City, Dr. Podell earned his doctorate of medicine from the University of Buffalo, class of 1926, and came to Red Bank the following year to establish a practice in general medicine. He took graduate training in proctology and ultimately specialized in that field. Until his retirement in 1974, Dr. Podell had been affiliated with Monmouth Medical Center in Long Branch and Riverview Hospital in Red Bank where he had been a member of the medical board. During World War II he served in the medical department of the AUS. Dr. Podell was 75 years old at the time of his death.

Dr. B. Radzivanas, M.D.

We just have learned that Bronislavas Radzivanas, M.D., formerly a member of our Essex County component, died of a heart attack on December 1, 1978. Born in Lithuania, Dr. Radzivanas received his medical degree from the University of Vytautas the Great in that country in 1939. Before emigrating to the United States he practiced medicine in Lithuania and Germany. Dr. Radzivanas became a citizen of the United States in 1955, took graduate work in radiology and practiced general medicine with emphasis on that specialty in Kearny. Prior to his retirement in 1975, Dr. Radzivanas was on the staff at West Hudson Hospital in that city. He was 71 years old at the time of his death.

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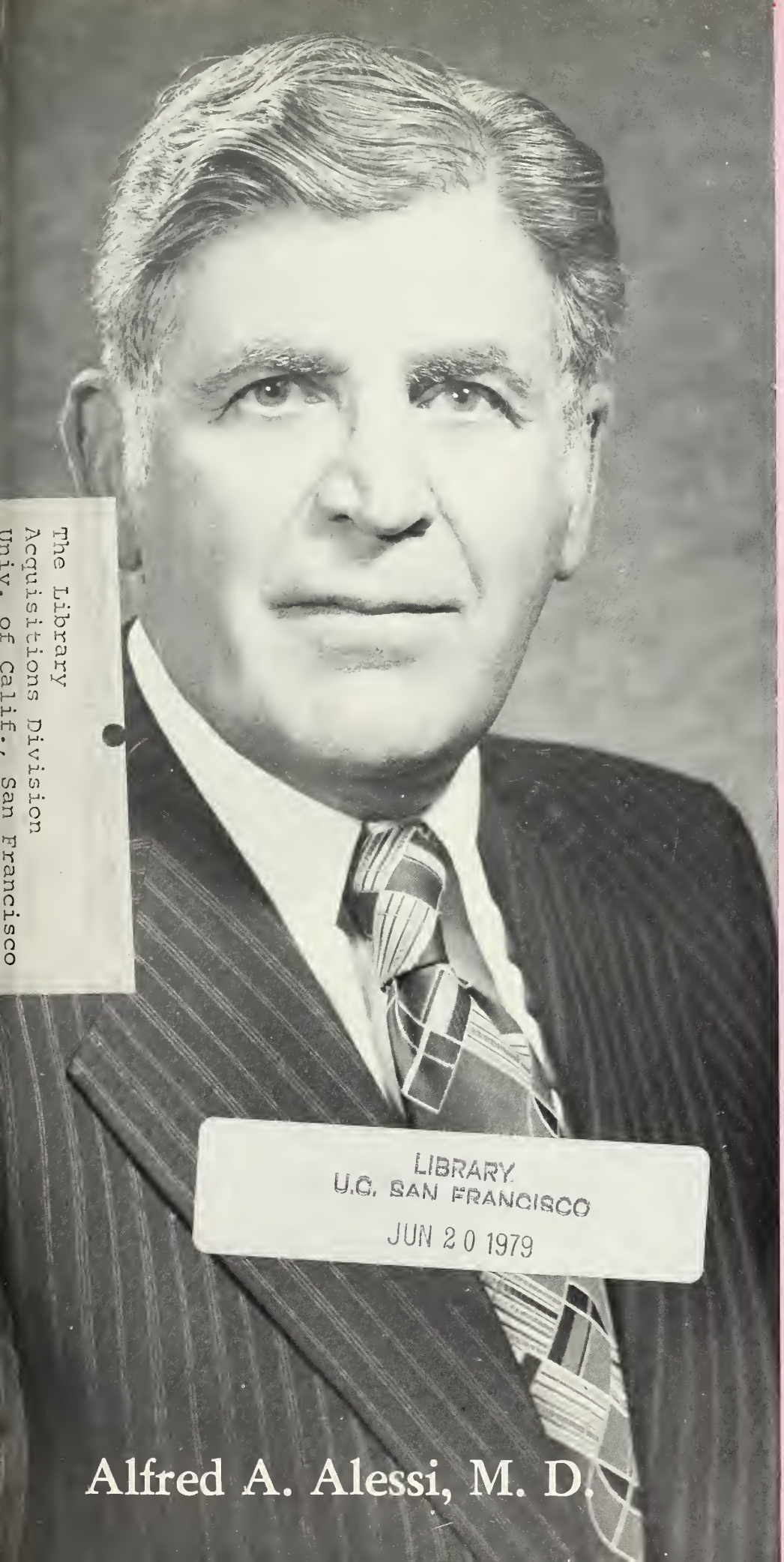
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Alfred A. Alessi, M.D., 187th President of the Medical Society of New Jersey, assumed the office on May 13, 1979. Dr. Alessi is a vascular surgeon with offices in Hackensack. (See editorial page 411, this issue.)

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Number 6

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The Maker

Examining a Few Myths About Prescribing.



Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand-name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.

MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were not bioequivalent to a reference product. As yo know, there is substanti literature on this subject affecting many drugs, in cluding such antibiotics as tetracycline and erythromycin. The record o drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of th generics. Drug nomenclature is not the important point; it's the compe tence of the manufacturer and the integrity of the product that count.

Matters.

MYTH: Generic options almost always exist.

FACT: About 55 percent of prescription drug expenditure is for single-source drugs. This means, of course, that for only 45 percent of such expenditure, is a generic prescribing option available.

MYTH: Generic prescriptions are filled with inexpensive generics, thus saving consumers large sums of money.

FACT: Market data show that you invariably prescribe—and pharmacists dispense—both brand and generically labeled products from known and trusted sources, in the best interest of patients. In most cases the patient receives a proven brand product. Savings from voluntary or mandated generic prescribing are grossly exaggerated.

MYTH: Drugs account for a major portion of the rise in health care costs.

FACT: Drugs represent a very small part of such costs. The amount of the health care dollar spent for prescription drugs was about 12 cents in 1967; today it is about 8 cents. And you as a physician are most conscious of how drug therapy can cut hospitalization, avert surgery, reduce office visits and keep patients on the job.

MYTH: Government intrusions into the marketplace will save tax money.

FACT: Government schemes always cost the taxpayer something, and the costs often exceed the benefits. Certainly, any federal "help," such as lists of wholesale drug prices sent to all physicians and pharmacists, will be no exception. Just think of the expense of keeping them current! Moreover, wholesale prices are poor guides to actual transaction prices and even worse guides to retail prices.

The PMA Position

We believe your freedom to prescribe, either by generic or brand name, should be totally unabridged. Otherwise, your prescribing prerogatives and your relationships with patients will be seriously impaired.

The maker does matter

After the myths about price and equivalency have been shattered, one fact stands out more clearly than ever: *The maker does matter.* As always, your best guide to drug therapy for your patients is to select products—both brands and generics—from manufacturers with credentials and performance records you have come to respect.

PMA

Pharmaceutical Manufacturers Association
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Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in postsympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

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CONTRAINDICATIONS: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components. Do not use in the eyes or in the external ear canal if the eardrum is perforated.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

EDITORIALS

Alfred A. Alessi, M.D.

Alfred A. Alessi, a vascular surgeon from Oradell, New Jersey, is the 187th President of the Medical Society of New Jersey.

Born in New York City, Al grew up in Rutherford, where he was graduated from high school. He received his premedical education at Colgate University, his medical degree at the New York University College of Medicine in 1938, and, in 1973, he was granted an M.S. from Fairleigh Dickinson University.

Dr. Alessi served internships and surgical residencies at the City Hospital, New York between 1938 and 1942. During the period 1942 to 1945, he was in the Army of the United States at the 14th Evacuation Hospital, CBI Theater in India, where he attained the rank of major in the medical department and was decorated with the Bronze Star for Meritorious Service.

Al married Isabel Molinari and they have four children—Joan, Alfred, Louise, and Thomas, a student at CMDNJ. By now they are grandparents for the seventh time.

Life has been a mixture of surgical practice and service for Al Alessi. In addition to being the Director of Surgery and Chief of Vascular Surgery at the Hackensack Hospital, he has served as a chairman of numerous committees of that hospital and as a member of its Committee of Directors and its Board of Governors. His service to the Medical Society of New Jersey and the Bergen County Medical Society has been tireless. He was a delegate from his county society for 15 years, and served in numerous other positions until he became its president in 1974-1975. Dr. Alessi takes over the leadership of MSNJ after three years in the presidential chairs—second vice-president, first vice-president, and president-elect.

Al has been a President of the American College of Surgeons, New Jersey Chapter, a member of the New Jersey Regional Medical Program, and numerous national vascular societies. He is a Diplomate of the American Board of Surgeons and a Fellow of the American College of Surgeons. He also holds fellowships in the New York Academy of Medicine and the Academy of Medicine of New Jersey. In 1964, he was named citizen of the year in the Hackensack Chapter, UNICO National.

Alone or with physician colleagues, Dr. Alessi has authored eight papers on surgical topics. He is Clinical Professor of Surgery at New Jersey Medical School, CMDNJ, and Adjunct Professor of Human Physiology at Fairleigh Dickinson University.

Despite all of the professional duties of his life, Al has had time for his family with whom he shares his hobbies of power boating, deep-sea fishing, swimming, skiing, and hunting. Both his wife and daughter Joan are involved in his surgical office practice. Al and his wife love music and regularly attend the Metropolitan Opera performances in New York.

The Medical Society of New Jersey is most fortunate to have Alfred A. Alessi as its President. We wish him the very best of luck and happiness in this responsible position.

A.K.

The Johnson Foundation and Student Aid

The Robert Wood Johnson Foundation of Princeton, New Jersey "is an independent philanthropy interested in improving health care in the United States." In 1972, the Foundation embarked on a nationwide program of financial aid for students enrolled in medicine and osteopathy. Between 1972 and 1976 $10 million was distributed directly to educational institutions for scholarships and loans to minority students, to women students, and to students from rural areas.

In its 1978 annual report, David E. Rogers, M.D., President of the Foundation, summarized an evaluation of the student aid program.*

More than 50 percent of the funds were used to enroll and maintain minority students—predominantly black students—in medical school. A corollary to this distribution figure was data from the USPHS National Ambulatory Medical Care Survey, which showed that black physicians provide office medical care almost exclusively to black patients. The study showed that 87 percent of the office clientele of black physicians were black and 10.5 percent were white. As one might guess 90.4 percent of the office patients of white physicians were white and 7.4 percent were black. The study also showed that black medical graduates were 26 percent more likely to choose primary care—family practice, internal medicine, or pediatrics—as a career as compared to white medical graduates. Furthermore, there was evidence that black physicians trained in the South tended to settle in that area and that a larger percentage locate in large cities where minority groups predominate.

*Annual Report 1978. Princeton, The Robert Wood Johnson Foundation, 1978, pp 9-14.

The data on women might have been guessed correctly: more go into pediatrics and fewer into family or general practice. They tend to cluster into hospital-based and salaried jobs, and into primary care specialties. As to physicians from rural America the data are quite impressive. Over 30 percent of physicians with roots in towns of less than 10,000 population settle into rural practice. This represents a rate three times higher than that observed among physicians who originate in urban areas.

From these data, among other conclusions, the report states that:

"1. An increased supply of black physicians can be viewed as helping to improve the geographic distribution of physicians.

"2. If medical care is to be equitably accessible to all, enlarging the number of black physicians is clearly of critical importance to the welfare of the nation.

"3. Targeting on particular groups of students can only modestly—not dramatically—change the selection of career and practice location."

One cannot fault this kind of philanthropy because the ever-increasing costs of medical education will make financial aid through loans and scholarships more important. As pointed out by Dr. Rogers, the federal fiscal policy regarding student aid has been rather fickle over the past decade, with emphasis first on ability, later on financial need, and most recently on student aid based on postgraduate service commitments. This confirms the need for private, industrial foundation, and other non-governmental fiscal aid.

The principle of preserving segregated office medical care —which the data clearly demonstrate—should be repugnant to all, especially to the black physician. Yet pragmatism dictates that social change is so slow that black patients cannot wait for the phenomenon of true integration in office practice to occur. Thus, an absolute increase in the number of black physicians would appear to be the pathway to office health care for our black citizens for some decades to come.

The Robert Wood Johnson Foundation should be congratulated for its good works in the service of New Jersey and the nation. A.K.

Are No Ethics Sacred?

Your editor recently listened to an unpleasant anecdote from a physician colleague. The resultant feeling of revulsion prompted this editorial. It seems that the physician's father, an elderly gentleman, was admitted to a hospital in a nearby state for treatment of a critical illness. Six physicians participated in the treatment program; the patient died nevertheless. My young associate was "surprised" when every one of the physicians on the case refused the Medicare assignment and insisted on full and direct payment. The physician questioned me as to the ethics of the situation. My response was that I was not "surprised"—I was shocked! To me, it was and is a disgrace.

Have we reached the point where the ethical canons on which those of us in our middle and older years were reared are now to be abandoned? Is this a byproduct of the new economics of medicine? Was this a peculiar situation related to a small group of physicians who refused to participate in a government-reimbursement program or was this just a case of extreme bad manners? One must hope it was the latter.

The medical profession always has "looked after its own." The basic concepts of courtesy, which should not need reiteration to any physician, are in danger:

1. Parents of a physician should be treated without direct fee.
2. The spouse of a physician should be immune from charges.
3. The dependent children of a physician should be treated without fee.

There is nothing unethical about collecting fees from third party payers who may represent such individuals, and this would include Medicare, Blue Shield, or a private insurance company. Doing so will not compromise the treating physician's principles, yet will preserve a very sacred concept.

Extension of "courtesy fees" to others, including allied health professionals and the clergy, is entirely a matter of personal dictums. Each physician has his own ideas on this matter.

In these times, we need all the self-respect and mutual respect we can garner to protect our public image which is being tarnished by outside forces. We must not now permit our ethical conduct to be impugned for reasons such as those described. A moment of careful thought will underline the wisdom of this approach. A.K.

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Let Us Face Our Problems*

ALFRED A. ALESSI, M.D., Hackensack

Our greatest danger is government intervention. The first step in solving our problems is to define them clearly. We must talk to patients, explain the problems, and solicit their support.

With the exception of my family and personal friends, many of whom are present today, my greatest pleasure in life has been the association with my colleagues in the Medical Society of New Jersey. I wish to extend my gratitude to all of you whose support, encouragement and, yes, votes have made it possible for me to stand here today.

In my view, the very essence of the care we render to our patients is that at all times we must tell the truth. There is no need for me to tell you that our system of medical care and medical education is in serious danger. Why is this so? The public hears that the treatment of catastrophic illness, and indeed, ordinary day-to-day health care, are being priced beyond its ability to pay. The concern is not only with the individual's ability to pay for health care but the government's continuing ability to bear its share of the cost.

This is hard to rationalize. As Peter Drucker so aptly pointed out in a recent article in *The Wall Street Journal,* almost 90 percent of the population does not pay for the medical services it receives. This cost is borne by some form of prepayment health plan. The majority of our patients are enrolled in employment-based plans and many others are on Medicare and Medicaid. Mr. Drucker points out that most patients neither know nor care what their individual health care insurance costs.

The press and TV have taken up the Kennedy cry of the need for National Health Insurance but we hear surprisingly little from the public demanding such National Health Insurance. I have to agree with Mr. Drucker when he says that he suspects the only reason we still hear of a demand for National Health Service is Senator Kennedy's understandable desire to have his name on one piece of major national legislation.

We all should become aware of what percentage of the national budget is given over to health care. We all should be aware of its relationship to the budgetary elements of defense, energy, housing, and general welfare, among other things. A decision has to be made by the public, politicians, doctors, and scientists together as to what percentage is necessary for health care and this may vary according to current needs. Our major concern is the government's encroachment through regulation of the delivery and access to medical care. We must bend all our efforts to retaining the good points of our health care system and to bring this about we must make the public aware of what it is that Senator Kennedy and Mr. Califano are about. How should we do this?

The AMA has retained several top public relations counselling firms to analyze the problem. The Gallup organization and others have underlined the problems but they have not solved them. That remains for us. The challenge is to change public attitudes. The only way public attitude is going to be changed in our favor is through day-to-day physician

*Inaugural address on the occasion of the author's induction into the presidency of the Medical Society of New Jersey. Presented to the House of Delegates, May 13, 1979, Atlantic City.

ontact with his patients. We must urge every physician to ake this as part of his mission—to spend an extra few ninutes with each patient, to talk to the patients, to explain he problem, and to solicit their support. Urge them to keep heir eyes and minds open and to recognize National Health nsurance propaganda for what it is. Any campaign we embark upon to change public opinion must not be self-erving. We must first of all believe in our hearts that the kind of care we render is the best care for our patients.

The first meaning of the word "doctor" is derived from the Latin "docere"—to draw out, to teach. We were teachers before we were healers. In addition to educating or drawing out our patients, we also should teach our young physicians and our medical students by example as well as precept. We should remind them that the first requisites of a young physician are the willingness and the desire to work, which are more important than intellectual genius.

Many of our members teach in medical schools. They should be clinicians with a great interest in patient care as well as teaching, and we should acknowledge that teaching ability is far more important than publication.

Let me break off from the main topic for the moment and tell you of a concern of mine. I address it specifically to you Delegates and to all those physicians whom we see face-to-face all too seldom. In Bergen County and here at the State Medical Society I have been involved closely with your officers and your Board for more than two decades. One of our problems is that we talk to each other and we fear that some of our decisions do not reflect "grass-roots" opinions. When you go back to your home societies, please stress that we want *your* opinions, we want our decisions to be based on what you want. I urge you to think carefully, to examine your own feelings, and to make sure that your officers and your Board are apprized of just what those "grass-roots" opinions are.

We have become aware over the years that close to the base of many of our problems regarding hospital costs is the fact that physicians are not involved enough in hospital investments. I suspect that if physicians were involved directly in hospital investment and, as they say, "owned a piece of the action," the physicians would demand that the hospital begin to function more like a sound business. I ask you to consider what would be the result in a given area if there were another hospital owned by physicians. Healthy competition would develop between the two and the patients and the people responsible for paying the cost of these operations would begin to share in the financial benefits as well as the quality of the care provided.

When you analyze statements by concerned people in the business world who have studied the medical care cost problem—and I refer to Peter Drucker, Harry Schwartz, and the editorial writers of *The Wall Street Journal*—one of the problems is that a great many of our corporate employees are accustomed to getting tax-free benefits as part of their fringe benefit package from their employer. They are thus completely unconscious of the cost involved. If we are to make the worker cost conscious we must find a way to make him aware of what those costs really are. The way medical costs are paid now, lay people make decisions and are completely unaware of the effect that their decisions have on the cost and indeed on the quality of the care we render. If these non-medical people continue to make decisions as to what care can and cannot be paid for, they will stifle innovative developments and soon we will be rendering cookbook medicine.

Another concern is our health planning system. Let me give you a "for instance." Hackensack Hospital recently paid over a million dollars to consultants to comply with regulations instituted by a variety of federal and state agencies charged with overseeing that new hospital construction complied with so-called cost-cutting programs.

I issue a warning for all of us: in medicine, change is a fact of life. I remind you that not every change is a step toward socialized medicine, as some of our own members would have us believe. To say that all change is bad is a blind assumption which keeps us from recognizing important health care improvements when they come. If we are to avoid the extension of the deadening hand of government we must assure our patients and the politicians that we are providing the best medical services in an atmosphere of free market principles at a price that our citizens can afford.

At the bedrock of everything I have to say is that our greatest danger is government intervention. We have been challenged by physicians who will say, "Why should I join the Medical Society of New Jersey? What has organized medicine done for me?" Some of us may say that's a naive question. Naive it may be, but it is a fair question and deserves an honest answer. The Medical Society of New Jersey and its component county societies protect the interest of all physicians practicing in this state. For example, in the offices of our county, state, and national associations we receive legislative newsletters every week. These staffs analyze all of the proposed legislation having to do with medical problems. I can assure you that these bills number in the thousands. Some of these represent serious threats, some merely are annoying, while others are downright silly and are aimed only at getting free publicity for the author of the bill. Silly, frivolous, or whatever, these must be addressed. There is no way an individual physician can defend himself against these threats. It takes a dedicated, informed, and hard-working professional staff at all levels to see that these threats are blunted. Without our professional organization to protect your interests, it would be difficult to practice in New Jersey and still be proud of your profession.

The key to everything that I have said today is that in order to halt government intervention at the county, state or national level we must enlist our patients to help us to change public opinion.

I have been warmed in the many hours I have spent with my colleagues at the State Society by the presence of our medical students. They are a refreshing lot and their enthusiasm, their intelligence, and their interest bode well for the future. Many of them are here in this hall with us and I want to assure them that I speak for all of us when I say they are most welcome. You are our hope for the future.

One of the great controversies of the last few years at the state society has been our efforts in continuing medical education. Though we have gotten brickbats from some quarters, we are going to continue to demand the high standards that our CME Program bespeaks. In the long run we all will benefit from our CME efforts, which are well worth the time and money we invest. Keep in mind one simple but important point: this is a demand we make on ourselves and it gives evidence to our critics that we not only are keeping up, we are keeping ahead of all other professions.

Hospitals conduct audits and develop criteria of patient care based on these audits. Sometimes we don't know how to evaluate these audits in terms of effectiveness of the care rendered to our patients. I'm afraid I don't have the answer to this, but in the coming years we must find the answer.

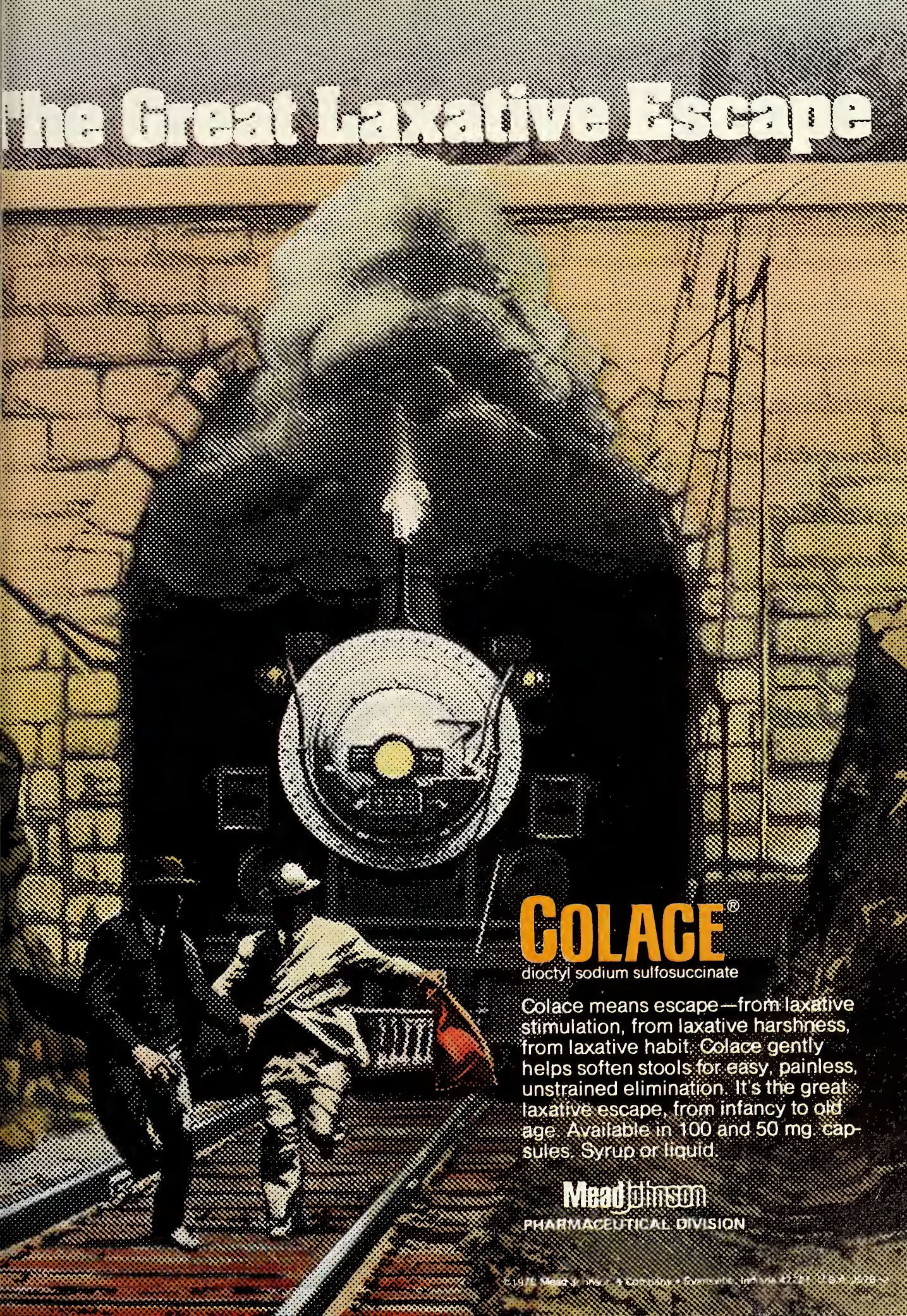
Let's prove, if we can, that CME and hospital audit can be translated into better care for our patients. Why do we worry about CME? Do we want to get the government off our backs? Or do we involve ourselves in CME because we want to present a nice shiny image to the public? Or do we involve ourselves in CME because we want to be able to render better care to our people? I say to you we do it because we want to help our patients. Now I say prove it! If we can prove that CME can be translated into better care for our people we may convince those few members who have dropped out of this Society because of CME requirements that they were wrong and that they should come back to the fold.

As I see it, the CME problem is that we lack scientific methods of measurement. Let me give you a concrete example. I think you will agree that many of our colleagues in practice ten years or more would have to sweat to pass examinations taken by some of those medical students who are with us today. Perhaps some would even fail. Yet, if you needed a doctor, to whom would you turn? I assure you your colleague would be your choice. You would have to go with experience. We have to take a second look at what we are doing with medical education.

One thing we don't lack is problems. They are all around us. We are attacking the physician's assistant problem, the emergency medical care problem, the national problem of nuclear energy, and national health insurance; PSROs we have always with us and Health Services Agencies dog our every step. We constantly are aware of our relations with the Medical School, and, in particular, what to do about a medical school in the southern part of the State. The problems go on and on. Right now I am preparing specific recommendations to present to the Board of Trustees addressing these problems at the earliest possible moment.

It has been reported to me that some of you attended a meeting in Philadelphia recently when Mr. I.C. Glendenning, President of the Federation of Telephone Workers, stated, and I quote precisely, "America's greatest sickness is its health care delivery system." You may tell Mr. Glendenning for me, "Baloney." Every time the Gallup people tested public awareness of their problems, medical care was well down the list. I will say to Mr. Glendenning, and any other critic of medicine, that the number one problem in this country is inflation. President Carter has been completely ineffective in dealing with inflation. In fact, every time he tries to meddle with the system it gets worse. And I say here and now the Administration is attempting to blame double digit inflation and its attendant high medical cost on the medical profession in an effort to divert attention away from itself and its failures. We must use every medium of public utterance to drive this point home.

As was made evident in those Gallup Polls and others that I have referred to, the first step in solving our problems is to define the problems clearly. Once we have done that we can roll up our sleeves and work together to find the answers. I am honored to have this opportunity to share my thoughts with you. You have won the gratitude of myself and my family. Thank you.


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Dosage and Administration: Oral: 10 to 20 mg., three or four times daily. Intramuscular: 5 to 10 mg.(1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Parenteral administration is not recommended in the presence of hypotension or tachycardia.

Intravenous administration should not be given because of increased likelihood of side effects.

Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted.

Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

Supplied: Tablets, 10 mg., bottles of 100, 1000, 5000 and Unit Dose; Tablets, 20 mg., bottles of 100, 500, 1000, 5000 and Unit Dose; Injection, 10 mg. per 2 ml. ampul, box of six 2 ml. ampuls.

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20 mg q.i.d. recommended dosage

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Precautions: Use with caution in patients with cardiac disease, hepatic or renal impairment. Concurrent administration with certain antibiotics, i.e., clindamycin, erythromycin, troleandomycin, may result in higher serum levels of theophylline. Plasma prothrombin and factor V may increase, but any clinical effect is likely to be small. Metabolites of guaifenesin may contribute to increased urinary 5-hydroxyindoleacetic acid readings, when determined with nitrosonaphthol reagent. Safe use in pregnancy has not been established. Use in case of pregnancy only when clearly needed.

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Long-Standing Pleural Effusions Remaining After Terminated Therapeutic Pneumothorax: Six Representative Cases*

PAUL SLAVIN, M.D., Newark

Long-standing pleural effusions remaining after terminated therapeutic pneumothorax generally are free from reactions. Attempts at aspiration of fluid may cause a dangerous perforation of the lung. Draining of the effusion is purposeless, since it reaccumulates promptly, proving its space-filling function. Intervention is needed only in rarely occurring reinfection of the pleura.

The use of artificial pneumothorax in the treatment of pulmonary tuberculosis mostly was discontinued in 1952 when isoniazide (INH) was added to chemotherapy of the disease. The majority of surviving patients who were placed on therapeutic pneumothorax prior to the establishment of antituberculosis chemotherapy are in the 50 to 65-year age range and are engaged in normal occupations. Many of these patients have residual long-standing pleural effusions which are filling the spaces left vacant by incompletely reexpanded lungs. The localized fluid collections generally have remained free from reactions for many years and have not significantly affected the breathing capacity. Attempts at aspiration may cause perforation of the adherent lung; drainage of the effusion is purposeless since it promptly reaccumulates at the same site, proving its space-filling function. Intervention is needed only in rarely occurring reinfection of the pleura, manifested by toxic symptoms of an acute pleurisy and by enlargement of the effusion.

CASE REPORTS

Case 1—A 61-year-old cabinet serviceman, who worked nine years in coal mines, was hospitalized in April 1946 for pulmonary tuberculosis. A right-sided pneumothorax was started four days later, followed by a left-sided pneumothorax. An effusion developed on the right side and was aspirated repeatedly until July 1947 when the lung was allowed to reexpand. The patient was discharged after a two-year hospitalization and has been returning to the Veterans Administration Outpatient Clinic (VAOPC) for periodic chest examinations. A major part of the right lung was obscured by a localized effusion and by thickened pleura (Figure 1, August 20, 1956). A chest x-ray, 28 years after discharge from the hospital, showed partial resorption of the effusion (Figure 2, March 29, 1976). The next periodic examination revealed a spontaneous pneumothorax located in the upper portion of the right lung; the air space communicated with the effusion, both being encapsulated within adherent portions of the lung (Figure 3, August 25, 1976). Two days previously, the patient had felt a strain while carrying a heavy kitchen cabinet. His breathing was not affected and he had no cough or sputum. Aspiration was not attempted. Subsequent examinations showed a steady decrease of the air space. The last examination, seven months after the accident, revealed complete obliteration of the pneumothorax; the effusion remained unchanged (Figure 4, April, 1977). After resting at home for five weeks, the patient returned to work.

Case 2—A 51-year-old barber had been treated for pulmonary tuberculosis by a private physician since 1948. A left-sided pneumothorax was started in 1951, followed by a pneumolysis. A pleural effusion which was positive for

*From the Veterans Administration Outpatient Clinic Newark, New Jersey. Dr. Slavin may be addressed at 351 Broad Street, Newark, 07104



Figure 1—Case 1—Right-sided post-pneumothorax effusion of nine years' duration.



Figure 2—Case 1—Effusion partly resorbed, free from reactions for 29 years.



Figure 3—Case 1—Spontaneous pneumothorax localized in upper portion of the right lung, and communicating with the effusion.



Figure 4—Case 1—Pneumothorax space obliterated; the shape and size of the effusion appear to be the same as before the spontaneous pneumothorax.

tubercle bacilli developed one year later and was aspirated repeatedly; the sputum was free from tubercle bacilli. After three and a half years, the lung was allowed to reexpand. Periodic chest x-rays continued unchanged; the right lung was distended; the retracted left lung was obscured partly by the displaced mediastinum and was overshadowed laterally by a localized pleural effusion. The hemidiaphragm was elevated (Figure 5, February 21, 1973). When barber shop income dropped sharply in 1972, the patient became employed as a night maintenance man in a nursing home; the change to strenuous work did not affect his breathing capacity. The last examination, 23 years after termination of the pneumothorax, showed no changes in the space-filling effusion (Figure 6, June 13, 1977).

Case 3—A 62-year-old dentist was hospitalized in August 1937 because of pulmonary tuberculosis. A left-sided pneumothorax was started and four months later the patient returned home to continue refills by a private physician. He was hospitalized again in 1940 when a right-sided pneumothorax was added. He was discharged after eighteen months to continue on bilateral pneumothorax. Examination revealed a partial selective collapse of the right upper lobe; the inner aerated half of the left lung was diffusely adherent along the mediastinum while the outer part of the field was overshadowed by a hydropneumothorax. The effusion grew larger and had to be aspirated repeatedly. The right lung showed no active lesions and was allowed to reexpand in December 1942. Twenty-one months later, an active lesion causing hemoptysis developed in the right upper lobe. Pneumothorax was reestablished and within nine days the air space showed a tendency toward a selective collapse of the upper lobe; lung markings of an obliterative pleuritis were seen in the background of the left pneumothorax space (Figure 7, August 14, 1944). Treatments on the left side were terminated in February 1945; on the right side, refills were discontinued in June 1948. In 1952, the tuberculous process in the right upper lobe became reactivated again and was controlled readily by triple-drug chemotherapy. The patient has been free from serious illnesses for the last 26 years. His right lung is distended, the outer half of the left lung is obscured by a localized effusion encircled with calcific plaques, and the hemidiaphragm is elevated (Figure 8, March 28, 1977). Examination in June, 1978 showed no changes.

Case 4—In 1945 a 49-year-old real estate salesman received pneumothorax treatments of the left lung. His chest films remained unchanged for 27 years; the right lung was distended and the diaphragm was detached from the liver by an interposed portion of the colon. The retracted left lung was overshadowed laterally by a residual effusion; the diaphragm was elevated and tented (Figure 9, January 24, 1973). On July 24, 1973, the patient complained of acute pain in the left chest, fever, and dry cough. Examination showed increased density and enlargement of the effusion (Figure 10, July 24, 1973). He declined immediate hospitalization, but because of increased chest pain, difficult breathing, fever and diaphoresis during the following night, he was advised to enter East Orange Veterans Administration Hospital (EOVAH) directly. On examination, in a medical ward, he was informed that his chest films showed no recent changes; he was discharged after five days. His ailing continued, and he decided to reenter the hospital. He was admitted to a tuberculosis ward in October 1973 and was placed on chemotherapy. Tubercle bacilli were found in cultures of the effusion; a partial decortication was done, and a resulting pleurocutaneous fistula was treated until the draining ceased. The 77-year-old patient, afflicted with additional serious illnesses, recovered from the tuberculous empyema.

Case 5—A 55-year-old accountant was hospitalized in March, 1947 because of hemoptysis. A right-sided pneumothorax was induced; he was discharged after thirteen months and continued refills as an outpatient. A pleural effusion developed in April 1949; treatments were discontinued in October 1950. His serial chest films covering 25

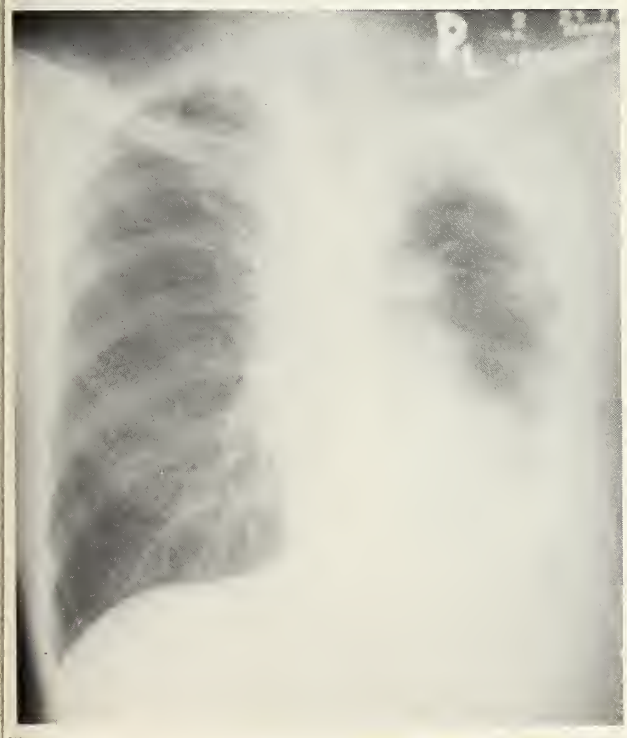


Figure 5—Case 2—Left-sided post-pneumothorax effusion of 19 years' duration.

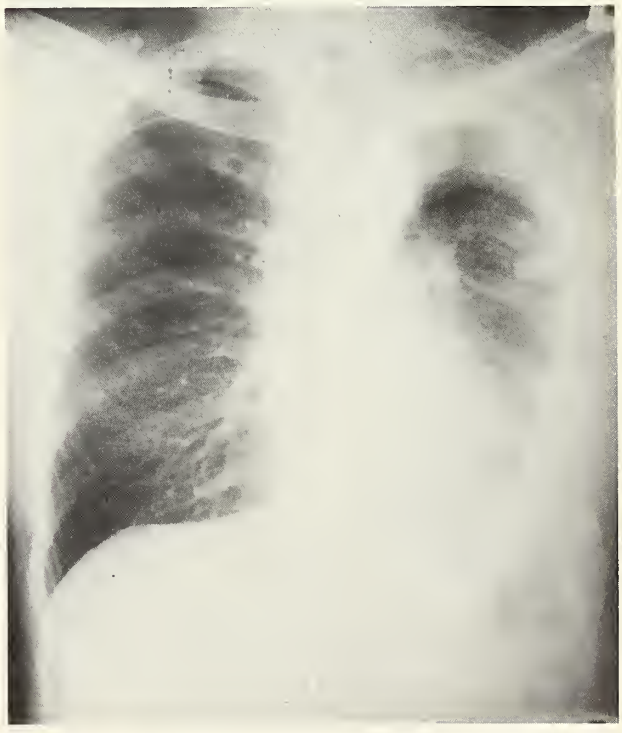


Figure 6—Case 2—Effusion has remained stationary for 23 years.



Figure 7—Case 3—Right-sided pneumothorax with selective collapse of the upper lobe. Left-sided hydropneumothorax overshadowing an obliterative pleuritis.



Figure 8—Case 3—Reexpanded right lung. Incompletely reexpanded left lung; partly resorbed effusion, free from reactions for 32 years.



Figure 9—Case 4—Interposition of colon. Left-sided post-pneumothorax effusion has remained stationary for 27 years.



Figure 10—Case 4—Recent enlargement and increased density of the effusion, signifying reinfection of the pleura.



Figure 11—Case 5—Right-sided post-pneumothorax effusion, stationary for 25 years.

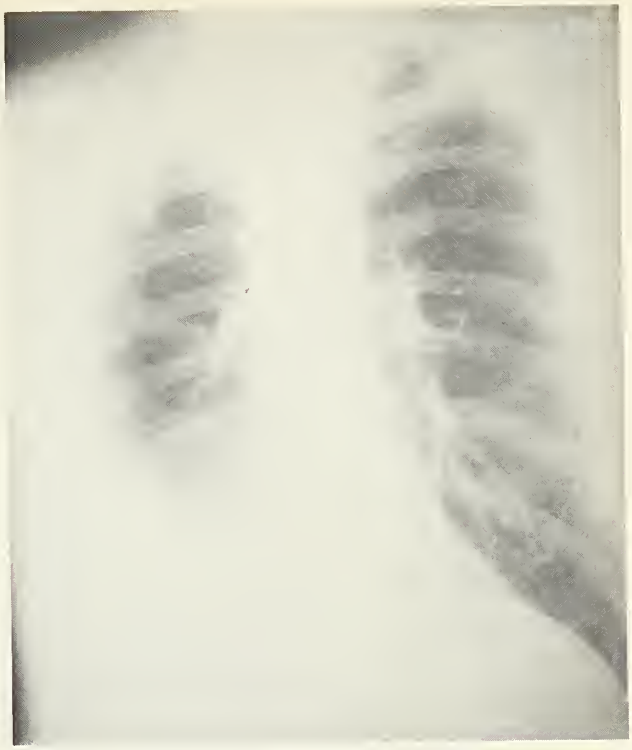


Figure 12—Case 5—Effusion reaccumulated and became positive for tubercle bacilli following a traumatic pneumothorax caused by aspiration.

years remained unchanged; the retracted right lung was overshadowed laterally by a localized effusion; the left lung was distented (Figure 11, July 14, 1975). The effusion was free from reactions and the sputum was negative for tubercle bacilli. On examination in January, 1976, the patient reported that in October, 1975 he was admitted to a private hospital because of an abdominal distress; his pleural effusion was drained, medication for tuberculosis, and treatments by a psychiatrist were started. The chest film at the clinic showed no changes in the dimensions and site of the effusion. The next examination six months later still failed to disclose changes in the appearance of the effusion (Figure 12, July 19, 1976).

In November 1976, the private physician advised a thoracoplasty because recent tests revealed the presence of tubercle bacilli in cultures of the effusion. The patient refused surgery. After reviewing the records of the patient's hospitalization, physicians at EOVAH stated that surgery was not indicated. The four-page summary submitted by the physician disclosed that on October 12, 1975, the patient was examined regarding an abdominal distress; noting the presence of a pleural effusion, the physician aspirated some fluid and in the process perforated the lung, causing a traumatic pneumothorax. The patient was hospitalized. Pulmonary function testing was not done before the aspiration nor during the stay at the hospital. The traumatic pneumothorax apparently involved tearing of encapsulated lesions containing tubercle bacilli, since cultures of the fluid were negative at first, but specimens submitted three months later were positive for acid-fast bacilli. The milky fluid was high in cholesterol and negative for fat; a lymphangiogram showed no damage of the thoracic duct. The occurrence of cholesterol in long-standing serous effusions was reported in 1917.[3]

Case 6—A 57-year-old businessman has been treated for pulmonary tuberculosis by a private physician. A right-sided pneumothorax was started in August 1948, followed by two pneumolysis operations. The sputum became negative for tubercle bacilli seven months later. Refills were continued once every week for four and a half years, until the VA cancelled further authorization of treatments. Physicians at EOVAH advised decortication combined with thoracoplasty. The private physician contended that maintenance of the pneumothorax will prevent an effusion; refills were continued, paid for by the patient, but the intervals were gradually lengthened to more than six weeks. Less than 100 cc of air at each refill was sufficient to maintain the pneumothorax space. After sixteen years of treatments, the chest films showed a wide-spread obliterative pleuritis involving large adherent portions of the right lung. Breath sounds were heard over the entire right chest. After eight more years of continued refills, the atelectatic upper lobe was reduced to a narrow, band-like density situated above expanded clear portions of the middle and lower lobes, each of them diffusely adherent along the mediastinum. Additional reexpanded parts of the two lobes were overshadowed by a shallow air space; their presence was identified by visible lung markings. The left lung was distended (Figure 13, October 26, 1972). The pneumothorax refills, uninterrupted for 26 years, finally were stopped. A thoracotomy was performed, but the diffusely adherent lung apparently was found unsuitable for decortication. Three years after the thoracotomy, the chest films showed an incompletely reexpanded right lung, a sizable space-filling effusion, a shift of the upper mediastinum toward the site vacated by the atelectatic upper lobe, and an elevated diaphragm. The left lung was distended (Figure 14, May 18, 1977). The uncommonly prolonged maintenance of the therapeutic pneumothorax took place during the period of established antituberculosis chemotherapy.



Figure 13—Case 6—24 years of continued right-sided pneumothorax treatments. The atelectatic upper lobe has been reduced to a small, narrow density situated above the expanded, clear portions of the middle and lower lobes. Additional expansion of the two lobes is overshadowed by a shallow air space.



Figure 14—Case 6—Incompletely reexpanded right lung after years of continued air refills, followed by a thoracotomy. A new formed space-filling effusion.

DISCUSSION

Pleural Effusions in the Course of Therapeutic Pneumothorax—With the aid of artificial pneumothorax countless patients recovered from their sickness without bearing lasting external marks of a chest disorder. Unlike surgical collapse operations, pneumothorax left no revealing scars and no significant deformities impairing costal breathing. It did, however, bring on intrathoracic complications and space adjustments. Permanent pleural pathological changes developed in every treated patient.[2] Effusions appeared in more than half of the cases.[3] At Montefiore Hospital and its country sanatorium in New York, the incidence of demonstrable effusions was 65 percent.[4] Some of them were of infectious origin, while others were sterile and probably due to irritation of the pleura by the air intervening between the parietal and visceral layers and by the use of local anesthesia at each refill. Alterations in the blood circulation of the compressed lung could have contributed also to accumulation of fluid in the pleural cavity. Proper aseptic precautions and the omission of anesthesia at refills would have reduced the occurrence of effusions.

Origin and Function of Long-Standing Postpneumothorax Effusions—Termination of a therapeutic pneumothorax depended on the results, including sputum conversion, cessation of pleural reactions, and radiographic evidence of completed healing. In successful cases the duration of treatments averaged about four years; pleural thickening and adhesions commonly were produced and were developed considerably more in the presence of effusions. The pleural changes prevented adequate reexpansion of the scarred lu and in many instances lessened its ability fully to replace t absorbed pneumothorax. Rise of the hemidiaphragm a shifting of the mediastinum aided in reducing the size of t unoccupied space and in some cases completed the filling. the presence of a preexisting effusion, any remainder of t vacuum was filled by residual fluid; in the absence of effusion at termination of treatments, a newly formed colle tion of sterile fluid completed the filling. Localized with adherent, unyielding portions of the lung, some of the spac filling effusions may remain stationary indefinitely whi others resorb partly or completely, marking their previo limits by thickened pleura and frequently by formation calcific plaques. In maintaining the intrathoracic space a justments, the encapsulated fluid takes the place of maj surgical operations; it is not to be regarded as a form infection and a source of undetermined ailments.

In cases 1 to 5, the space-filling fluid was a remainder of preexisting effusion. In case 6, a newly formed collection sterile fluid filled the space left vacant by the incomplete reexpanded lung. Terminating the refills years earlier woul have resulted in a wider reexpansion of the middle and low lobes, less displacement of the mediastinum and diaphragn a smaller effusion; and avoidance of a thoracotomy.

Management—Asymptomatic long-standing post-pne mothorax effusions need no intervention. Individuals fo merly treated for pulmonary tuberculosis usually return fo periodic examinations. The new chest x-rays are investigate for possible changes in the size and shape of the effusio

which may occupy a shallow space overshadowing most of the lung field and simulating an extensive collapse of the lung by obscuring wide-spread reexpansion. On rare occasions, fluid may reappear at the site of a resorbed localized effusion; it causes no subjective symptoms and is resorbed shortly. It is preferable to treat the patient with prophylactic chemotherapy. A rare reinfection of the pleura, brought on by contributory factors, is revealed by toxic symptoms with simultaneous spreading of the effusion. It should be treated as a tuberculous empyema.

SUMMARY

The paper outlines the origin and behavior of long-standing effusions remaining after terminated pneumothorax. Six representative cases are discussed. The effusions have been free from reactions for over 30 years in case one, over 23 years in case two, and over 33 years in case three. In case four reinfection of the pleura developed after 27 years of stability. In case five, injudicious aspiration of a serous effusion, which remained stationary for 25 years, resulted in a traumatic pneumothorax; the fluid reaccumulated promptly and became positive for tubercle bacilli later on. In case six, a right-sided pneumothorax was maintained for 26 years with the intention of preventing an effusion. A thoracotomy finally was done, and a newly formed effusion filled the space left vacant by the incompletely reexpanded lung.

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Patients Who Kill Their Physician

EUGENE REVITCH, M.D., Edison*

Physicians, and particularly urologists, proctologists, plastic surgeons, psychiatrists, and possibly orthopedists performing spinal operations are vulnerable to homicidal attacks by their disgruntled patients whose motive is retribution for real or imaginary injustices and suffering caused by their physician. Paranoid projections and depression with hypochondriacal preoccupations are responsible. The case of a patient who killed his psychiatrist is reported.

The purpose of this paper is to warn physicians that they may become the target of homicidal assault by a disgruntled patient. Urologists and proctologists who deal with the genital organs and the anal orifice are the most likely to arouse suspicion and anxiety of paranoid patients. Retribution for real or imaginary injustices, allegedly perpetrated by the physician against the patient, is the leading motive. The physician may have treated the assailant for a number of years or may have had little direct or indirect contact. Heads of institutions or chiefs of services may become the primary target.

Two types of patients are likely to kill their physician: paranoid patients and depressives with vague, continuous hypochondriacal complaints. Paranoids head the list.

CASE PRESENTATION

Several years ago the author was asked to examine a 53-year-old man for sanity determination. The history indicated that he had entered, unscheduled, the office of his psychiatrist and sat patiently in the waiting room for about two hours while the physician was treating other patients. Finally when called into the office and told that it would be a short visit, he shot the doctor in the chest, killing him on the spot. Following the murder he surrendered to the police.

The murderer was the psychiatrist's patient for a period of 13 years for treatment of depression. During that time he had been hospitalized for electroshock therapy on three occasions. The killing took place within three to four weeks following the last hospitalization.

The patient was a machinist with high school education, who always supported his family. His own early life was marked by poverty with severe emotional and material deprivation. His father was an abusive alcoholic who eventually died in a mental hospital. The patient was of normal intelligence with a full scale I.Q. of 107 on the Wechsler-Bellevue Intelligence Test. Since the test was given within approximately two months following the last electroshock treatment, it is quite probable that he had a higher intellectual endowment because of reversible memory impairment usually caused by this therapy. Lack of confidence, depression, perfectionistic concerns, and looseness of thinking were elicited on the projective tests.

In the psychiatric interview the patient was cooperative and extremely appreciative of the attention given to him. He felt he needed help. His speech was coherent and logical. He was well oriented and he could give his life history and the course of events leading to the homicidal act with clarity and in good chronological order. There was no evidence of delusions, hallucinations, or paranoid thinking. He was known as a very accurate worker to a point that his employer once remarked that he was "too fussy" with his work. On this occasion, possibly because of low mood and the effect of

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electroshock therapy, his performance was criticized. Over the weekend he brooded over his "failure," and the day of the incident he resigned from his job over the objection of his employer. He then proceeded to the doctor's office.

He later said that he did not intend to kill the doctor, but just to have him suffer "as I suffered." The killing, he felt, was a "mistake." His account of his relationship with the psychiatrist was interspersed with description of incidents he interpreted as rejection and lack of interest. This, however, was always followed by some statement exonerating the doctor. He hoped for a closer relationship with his doctor and he added, "I expected more of him. He was really not concerned and I felt there was no personal interest." He spoke a great deal about the deprivation in his childhood and he recollected that he had never received money from his parents for ice cream. He also disclosed resentment against his father.[3] He believed that he would finish his own life as his father did, in a mental hospital.

The court considered the patient insane at the time he committed the offense and committed him to a mental hospital under the purview of the McNaughten Rule.

REVIEW OF LITERATURE

Very little has been written on the specific problem of patients killing their physician. Dupré[1] classified these patients as "persecuteś-persecuteurs" (persecuted persecutors) in a paper he presented to the Société-Médicopsychologique July 16, 1918 on the occasion of the murder of a Professor Pozzi by a patient on whom he had operated for varicocele. The patient committed suicide following the shooting of the surgeon while the latter was sitting in his office. In a review of the then available literature on the subject Dupré referred to the case reported by Legrand du Saulle in his 1877 treatise of legal medicine. The case is so illustrative that it deserves to be mentioned in full translation.

"A coachman named Bourgeois who had been treated by our colleague, accused Dr. Bleynie of mishandling his 'cold in the intestines.' He complained of an illness which resisted the treatment by Dr. Fièvé for sixteen years. This hypochondriac then consulted Dr. Bleynie, who ordered hot baths followed by bathing in the river. He then consulted Dr. Biett in the hospital St. Louis in Paris, and steam bath was suggested. The suffering of our insane increases and he then declares that the treatment had dessicated his intestines, and he accuses the physicians of torturing him and causing an incurable condition. He then decides on revenge and he kills Dr. Bleynie with two shots from his pistole."

Another typical case mentioned by Dupré was Dr. Guinard, a surgeon at the Hotel Dieu in Paris, who was killed by a patient on whom he had performed an operation for a "chronic affection of the anus." Alliez *et al.*[2] reported more recently the killing of a physician by a foreign worker employed in France. The physician had seen this patient only on one occasion. The patient failed to fill the doctor's prescription believing that it contained poison and that the victim of his assault planned to have him expelled from the country.

In the case when the homicidal patient attacks the physician in the emergency room or in his office, Frazier recommends the approach of "talking down an armed patient in violent crisis."[3] According to him confrontation in an open hallway rather than in a closed room is preferred. The "talking down" takes a long time and "it takes more staying power than bravery." Madden *et al.*[4] warn clinicians to be sensitive to the precursive signs of possible assault such as agitation, threatening verbalization, and gestures.

DISCUSSION

The murder of a physician by his patient is relatively rare, but such an eventuality cannot be discounted. Most of the murders and harassments of physicians are committed by their paranoid patients. Paranoid phenomena can range over a wide spectrum of clinical conditions from major paranoid psychosis to a paranoid syndrome secondary to the psychogenic and organic conditions.[5] Paranoid personalities are prime candidates for dissatisfaction with medical care. Procedures involving the genitalia and excretory system may lead to malpractice action or to assault on the physician. A paranoid patient may become violent in the middle of a sigmoidoscopic examination.[6] Not all the paranoids are overtly and recognizably psychotic. The paranoid projections may be interpreted by observers as true statements when they are not bizarre in content. Furthermore, they may be hidden behind a facade of adequacy at work and superficial social adjustment.[7]

The paranoid patient may nurture his grudge for many years and attack when his name or perhaps very existence has been forgotten by his victim. I recollect two such cases reported in the press, one about the time of World War II and the other one about 25 years ago. An orthopedist and a cardiologist who had been mistaken for the patient's psychiatrist, were the victims. Podolski[8] believes that the purpose of paranoid homicide is to discharge overwhelming rage. This reestablishes psychic homeostasis, helps to handle anxiety and to gain a feeling of omnipotence. As murder by a paranoid may be preceded by years of brooding, so also in malpractice suits the physician may be harassed and abused for years, particularly if the patient lost his case. This has been my experience with paranoid litigants.

Brancale observed in his rich experience that the litigation and harassment give vent to the accumulated rage, and this may prevent bodily assault.[9] This observation is probably correct, yet caution is recommended. The present climate of anti-intellectualism, anti-elitism and irrationality coupled with presentation of physicians, and particularly psychiatrists, as villains by the communication media and such films as "One Flew Over the Cuckoo's Nest," may abet pathological aggression and direct it against the physician. Murder of the physician is only one aspect of this aggression and release of rage. Feelings of helplessness, anxiety, rage, and frequently inferiority feed the paranoid's assaults. Experts on violence recommend that the potential victim acknowledge and show fear in case of a threat and refuse to take the assailant's weapon, even if he offers it. This relieves his rage and feeling of helplessness.

In the case presented by Alliez *et al.*[2] paranoid phenomena started following an accident at work for which the patient had been awarded 100 percent compensation. Diffuse hypochondriacal complaints with depression are common following accidents. Expectation of reward certainly influences the patient's attitude in most of the cases, but the main fabric of post-traumatic mental states is infantile regression and the emergence of anxiety and rage among other factors.[10] It is therefore conceivable that exaggerated expectations of reward and paranoid projections and elaborations may be stimulated and released in predisposed individuals following trauma. It is my opinion that the examinations and reexaminations by many physicians, the

depositions, a cold and businesslike approach by the examiners, and the whole adversary process surrounding the patient aggravate the condition and increase the possibility of violence. Suicide following murder, as in the case of the murderer of Professor Pozzi, is well documented.[11] In some cases the murderer even may demand capital punishment. Murder may follow suicidal preoccupations or may succeed a combination of suicidal and homicidal fantasies.[12] A recent case of murder-suicide in New Jersey was reported in the press.[13] A disgruntled worker who lost his job and was not reinstated, shot himself after killing the company's founder and the two foremen who had testified against him in court. It is conceivable that a physician's testimony against a patient's claim in court may induce a similar outcome. In some cases the future murderer notifies his physician, friends, or minister that he may kill a specific person. This should not be taken lightly.[14] Unfortunately, a patient's warning frequently is not taken seriously.[12]

A case in point is the well-known Tarasoff case in California. Tatiana Tarasoff, a student at Berkeley University, was killed by a boyfriend who actually warned his psychotherapist of his intent. The psychotherapist notified the police but the patient was not detained since "he looked normal." There is no preventive detention and patients committed to mental hospitals are discharged hastily through the "revolving door system." For this and other reasons there is little protection against a paranoid litigant or a "persecuted persecutor." It is, therefore, an obligation to inform the potential victim of a threat to his life revealed by a patient to his physician, whether the prospective victim is a colleague or anyone else. This revelation of a patient's confidence cannot be considered legally as a breach of confidence.

The patient presented here belongs to the group of depressives with vague, continuous hypochondriacal complaints, with no or perhaps with only fleeting paranoid phenomena. Murder and other crimes committed in depressive states were described by Woddis.[15] This, in my experience, is not uncommon. In the background of our patient's grudge was the severe material and emotional deprivation of his early life, an abusive and rejecting father and memories, still painful over four decades later, that he could not afford an ice cream cone in his childhood. A patient with a history of emotional deprivation and in need of support may interpret lack of immediate response by the physician as indifference and rejection. In fact many suicides occur following such perception of the physician's attitude. If the victim of this patient had greeted him warmly in the waiting room or had made some other sign of recognition and concern, he still might be alive. Telling the patient that he would have only a short visit further increased his feelings of rejection, indifference, and neglect. It is quite probable that the homicidal act was actually a transference reaction, the doctor paying with his life for the abuses of the patient's alcoholic father. Frazier mentions that some psychiatrists were victims of patients they had committed to a mental hospital and he advises to "do the patient the courtesy of a follow-up visit."[3] It is, however, difficult and at times impossible for a busy practitioner to be available immediately and to make visits to distant hospitals. Patients, however, appreciate such an availability and this may save the life of (or at least a malpractice action against) an unsuspecting physician.

SUMMARY

Killing of physicians by a patient is relatively rare. Perhaps for this reason literature on the subject is scant. Two types of patients are likely to commit homicidal assaults on their physicians: the paranoid and the depressives with chronic complaints. The paranoids head the list by far. Urologists and proctologists, because of their dealing with areas of sexual significance, psychiatrists, and possibly orthopedists are the main targets. It is suspected that legal involvement in a malpractice suit against the physician may absorb the paranoid patient's rage and thus deflect him from assault on the physician. It is suggested to acknowledge and show fear in case of an assault by a paranoid patient because this decreases the assailant's anxiety and feeling of helplessness. Ready availability to the patient will prevent not only many suicides, but also will prevent many malpractice suits and homicidal assaults.

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Sweat Gland Carcinoma*

DONALD ROTHMAN, M.D., and
NATALIO DAMIEN, M.D., Red Bank

This case report illustrates a typical sweat gland carcinoma in its appearance and behavior. Suggestions for therapy are presented.

Sweat gland carcinomas are not common lesions. There are numerous reports in the literature of small numbers of individual cases and a few larger series.[1-3] A growing consensus indicates that these growths should be treated like melanomas with wide local excision and regional node dissection where applicable. A recent case at Riverview Hospital here is reported.

CASE REPORT

A seventy-nine year old woman presented in October, 1977 with a two-year history of a right buttock lesion. She had neglected to obtain medical opinion despite its growth to a large size. There were no predisposing factors. She had hypertensive cardiovascular disease, compensated heart failure and was treated with digoxin. She had had no surgery.

On examination, the lesion was purple, raised, and smoothly nodular with irregular margins deeply infiltrating the underlying subcutaneous areas (Figure 1). It appeared to move over the gluteal muscles. There was a small right inguinal node, but no hepatomegaly was found. A preliminary biopsy was interpreted as a lesion of sweat gland origin—benign. Under general anesthesia the entire lesion was widely excised and closed primarily without tension. The area healed. The node continued to grow so it was excised with local anesthesia; it proved to be metastatic malignancy of sweat gland origin. In February 1978, the patient underwent a radical groin dissection; liver scan and routine studies were normal. There was no residual tumor. Recovery was delayed by some flap necrosis and digitalis intoxication. She was discharged after three weeks; the wound healed and she became ambulatory without edema of the lower extremity.

PATHOLOGY

Grossly, the tumor was somewhat polypoid and measured 5.5 x 3.5 x 2 cm. (Figure 1). The gray-brown surface was crusted and ulcerated. When cut, the tumor surface was firm, grey and white. Microscopically, the lesion was arranged in lobules surrounded by hyalinized tissue. Central necroses suggested pilar origin (Figure 2). Ductal differentiation, cyst formation, and mucin production make this most probably a sweat gland tumor of eccrine origin. Deeper nests of anaplastic cells indicated infiltration; many mitoses were present (Figure 3). The lymph node showed metastatic disease of the same type.

DISCUSSION

This group of neoplasms of sweat gland origin has confusing cell types because the precursor may be apocrine or eccrine and from different parts of the sweat gland apparatus. Our particular case appeared eccrine and originated on the buttock—an uncommon area for these lesions. Usual-

*This case report is from the departments of surgery and pathology, Riverview Hospital, Red Bank. Dr. Rothman may be addressed at 135 Maple Avenue, Red Bank, New Jersey 07701.



Figure 1—Gross buttock lesion showing margins, lobulation, red-purple color.

ly occurring in the elderly, these neoplasms affect men and women equally. The tumors are firm, nodular, raised, occasionally ulcerated and purple-red in color. The usual size is two to three centimeters but occasionally they enlarge to ten centimeters. Usually they are found on the scalp, upper limbs, axilla, and upper trunk. They are slow growing but can metastasize to nodes, skin, vertebra, and other organs.[4]

The histology is not uniform, can be well differentiated, glandular, ductular, papillary, or undifferentiated. They lie deep to the dermis and invade. They can appear benign and still metastasize. The metastases may be more solid or epidermoid. The lesion may be mistaken for a non-sweat gland metastases (such as of breast or cutaneous origin) if found in the axilla. The incidence of metastases is high and in one study, 29 of 68 developed positive nodes in five years.[3] Newer histochemical and electron microscopic methods may help identify the sweat gland cell type.[5,6] Not enough experience has been gained with chemotherapy, but it should be used if the disease is widespread. Sweat gland neoplasms have been shown to be radioresistent.[7]

In view of the above, the treatment should be aggressive with wide excision and lymph node dissection despite the benign appearance.

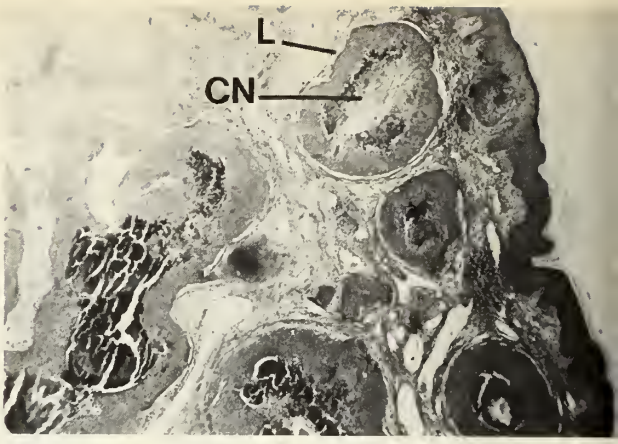


Figure 2—Low power view with many lobules, central necroses, and deep extension.

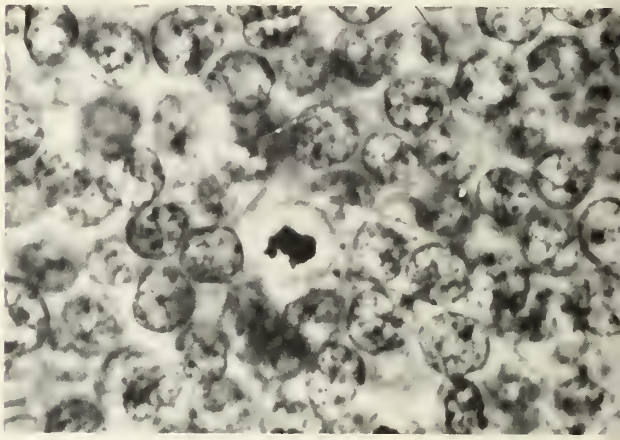


Figure 3—High power view of malignant cells and mitoses.

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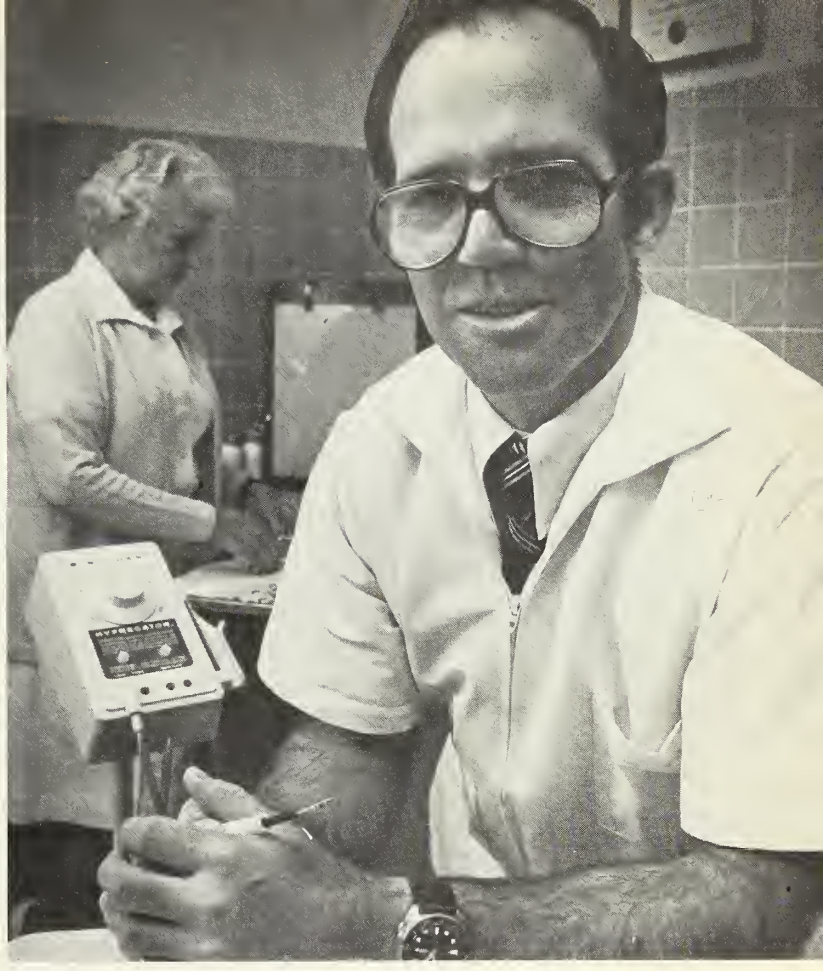
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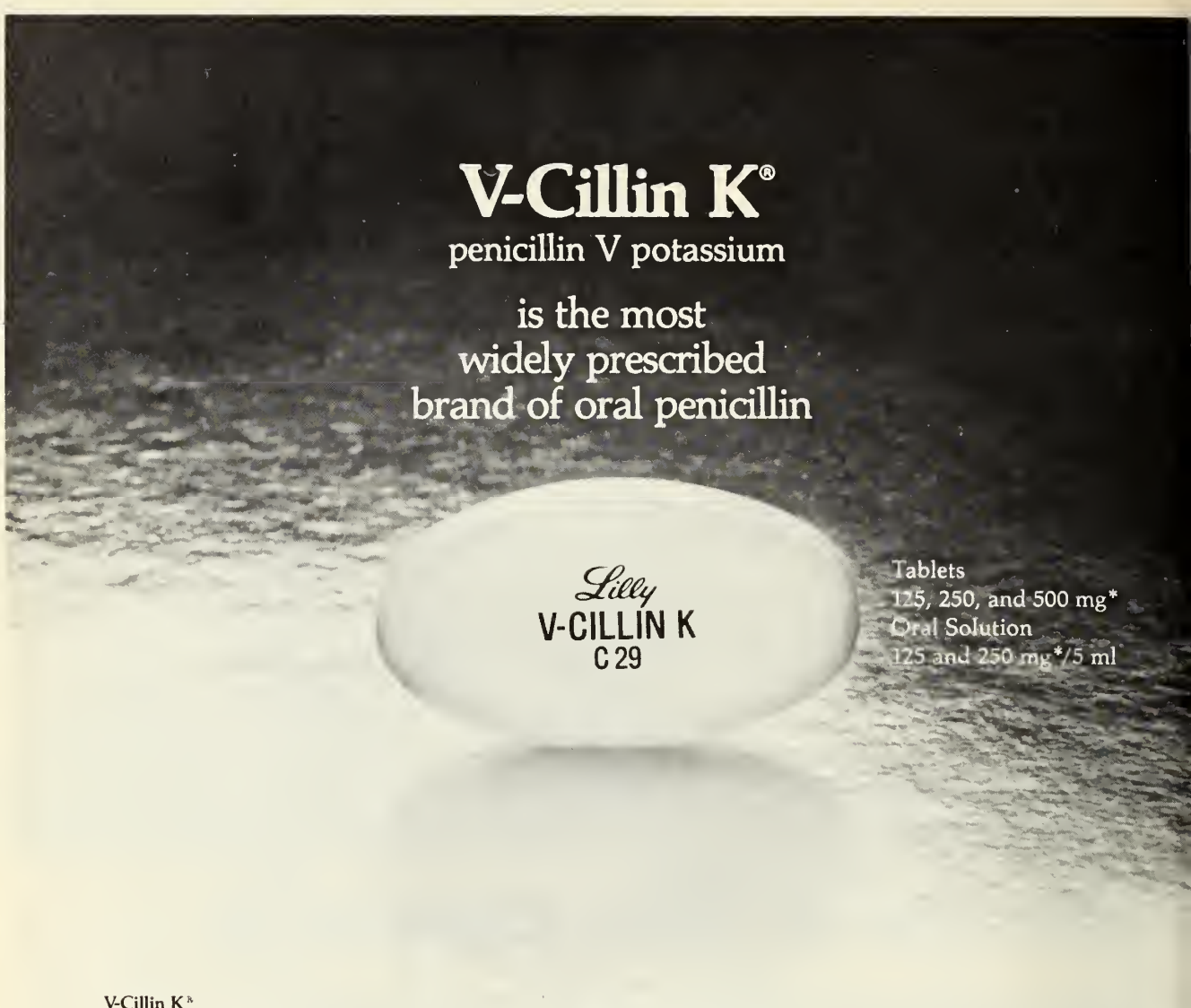
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Dissecting Aneurysm of the Internal Carotid Artery*

RADE PEJIC, M.D., Camden and
JOSEPH GRADY, M.D., Detroit

Dissecting aneurysm of the internal carotid artery is a rare lesion. It may occur spontaneously or be precipitated by head and neck trauma. However, an underlying disease process is usually present in the vessel wall, such as fibromuscular dysplasia or arteriosclerosis. Embolization to the brain is a constant threat and thus surgical excision with repair should be performed if the vessel is patent. Proximal ligation may be performed if the dissection extends intracranially and the carotid stump pressure is 65 mm Hg or greater.

The most common lesion affecting the carotid bifurcation and the internal carotid artery is a stenotic or ulcerating atheromatous plaque. Dissecting aneurysm of the internal carotid artery, a relatively rare pathologic entity, first appeared in the medical literature in the 1940's.[1,2] Since then, less than fifty cases have been reported.

In addition to involving the internal carotid artery, the dissecting aneurysm may extend into its major intracranial branches. It may occur without any obvious precipitating event.[3-8] More commonly, the dissecting aneurysm may be secondary to head and neck trauma[1,2] (as in our case), to carotid puncture for selective carotid angiography,[9,10,11] or be associated with a more proximal dissection from the aortic arch or common carotid artery.[12,13]

Whether the dissection occurs spontaneously or is precipitated by some traumatic episodes such as those described above, an underlying disease process usually is present in the wall of the vessel in question.

Our case report is somewhat unique in that the patient developed neurologic symptoms several months after the traumatic episode and transient ischemic attacks (T.I.A.s) approximately three and one-half years after his accident.

CASE REPORT

The patient was a 53-year old male who developed transient blindness of two hours' duration in his left eye three weeks prior to admission and again on January 11, 1978, one day prior to admission to Saint John Hospital. He denied any motor weakness in the upper or lower extremities, paresthesias of the face or arms, dizzy spells, or blackouts. The patient was not a diabetic and he smoked thirty cigarettes daily.

In June, 1974, while bicycle racing his son, the patient fell off his bicycle; his son's bicycle ran over the left side of his neck. Immediately after the accident, the patient noted only minor contusions and abrasions and felt reasonably well. Six months later, he developed a facial tic affecting the right eyelid and the muscles at the right angle of the mouth (orbicularis oculi, levator labii superioris, and levator anguli oris).

The vital signs and the entire physical examination, including the fundoscopic, were normal except for a right facial tic and bilateral soft carotid bruits (left greater than right). Chest x-ray, EKG, and all routine blood chemistry studies were normal. Aortic arch study (Figure 1) revealed a dilated and slightly tortuous left internal carotid artery. Two areas of constriction were seen on it (points A and B), which

*From the Department of Surgery at Saint John Hospital, Detroit, Michigan, where Dr. Pejic was a cardiovascular fellow. Currently he is on the surgical staff of the Cooper Medical Center, Camden, and the Garden State Community Hospital, Marlton. Joseph Grady, M.D., is Chief of Vascular and General Surgery at Saint John Hospital, Detroit, Michigan. Correspondence may be addressed to Dr. Pejic at 423 Cooper Street, Camden 08102.

represented the origin and reentry of the dissecting aneurysm.

At surgery, the left internal carotid artery was tortuous and measured 15mm in diameter proximally. A bluish discoloration was seen 12 mm from its origin which represented the entry point of the subadventitial dissection. This dissection involved approximately 70 percent of the circumference of the internal carotid artery. An 11 mm segment, which included the point of entry of the dissection, was resected. Inspection of the cross-section of the distal internal carotid artery revealed a two mm true lumen and a five mm false lumen which was formed by the dissecting hematoma. By passing a #4 Fogarty balloon catheter through the false lumen, the dissected intima and media, as well as the median septum, were removed completely and thus only one subadventitial lumen remained. A six mm Bakes dilator was passed with ease through the new channel toward the base of the skull. Thus an end-to-end anastomosis was performed between the proximal internal carotid artery and the distal false lumen, relying upon the point of reentry at the base of the skull to maintain circulation to the brain.

Microscopic examination of the specimen revealed fibrosis of the intima and media with extensive hemorrhage, fibrin deposition, and mural thrombosis within the wall of the vessel, a finding consistent with a dissecting aneurysm. No significant intimal arteriosclerosis was seen.

Postoperatively, the patient had no neurologic deficits and was discharged on the fourth postoperative day. Six weeks later, the patient was completely asymptomatic except for his right facial tic. Follow-up arteriogram (Figure 2) revealed no evidence of stenosis and good flow through the left internal carotid artery. At his six-month follow-up visit, the patient remains completely asymptomatic and is working at his previous place of employment.

DISCUSSION

Dissecting aneurysms of the internal carotid artery are usually secondary to an extrinsic or intrinsic precipitating factor. If the patient is hypertensive, dissection may occur even more readily.

Extrinsic precipitating factors may be head and neck trauma such as torsion or sudden extension or compression of the neck, proximal common carotid puncture for angiography, or extension of dissection originating at the aortic arch.[4] Intrinsic vascular lesions which may facilitate transmedial or subadventitial dissection of the internal carotid artery are mycotic, cystic medial necrosis, fibromuscular dysplasias (elastic and/or muscle tissue degeneration[3]), atherosclerotic, syphilitic, polyarteritis nodosa, giant cell arteritis, and granulomatous arteritis.[14,15] Thus, the term "spontaneous" dissecting aneurysm is somewhat confusing. It does not mean that the arterial wall is completely normal, but only implies that no clear-cut factors precipitating the dissection are apparent. There always is some type of abnormality affecting the media or the intima of the vessel.

Surgical intervention is indicated only in those patients who have a patent but stenotic internal carotid artery, as seen by angiography. If not repaired, subsequent embolization is a constant threat because of clotted blood present in the wall of either the true or false lumen of the affected vessel.[16] However, if the dissecting aneurysm cannot be excised or repaired because of distal intracranial extension, then the internal carotid artery may be ligated safely in order to prevent subsequent embolization, providing that the stump pressure is in excess of 65 mm Hg.[16] This level of stump pressure indicates very good collateral blood flow.

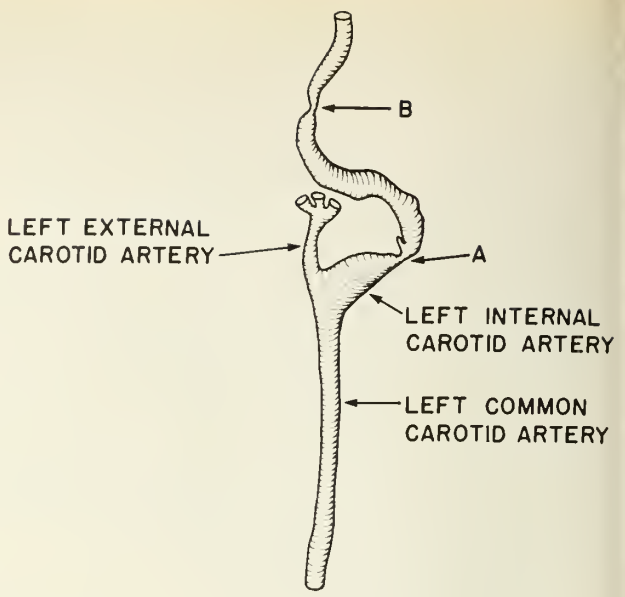


Figure 1—Schematic drawing of the preoperative arteriogram. (A) represents the origin of dissection and (B) is the reentry point in the internal carotid artery. This vessel is dilated and somewhat tortuous.

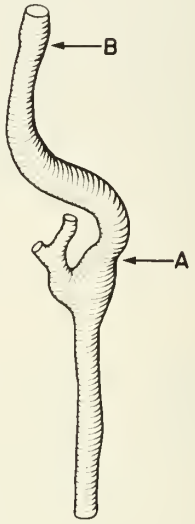


Figure 2—Schematic drawing of the postoperative arteriogram. (A) represents the point at which an end-to-end anastomosis was performed following resection of an 11 mm segment of the internal carotid artery. (B) is the point of the previous reentry channel.

SUMMARY

Dissecting internal carotid aneurysms may remain completely asymptomatic. When they become the source of cerebral embolization, they frequently are mistaken for an ulcerating internal carotid artery plaque. However, fibrous dysplasias, cystic medial necrosis and various types of arteritis are the usual underlying lesions rather than arteriosclerosis.

Angiography may be misleading and selective arteriograms are necessary to define the lesion precisely. However, the exact diagnosis still may not be made until surgery is performed and the bluish transmedial or subadventitial dissection is seen.

Surgical repair should be attempted if complete occlusion

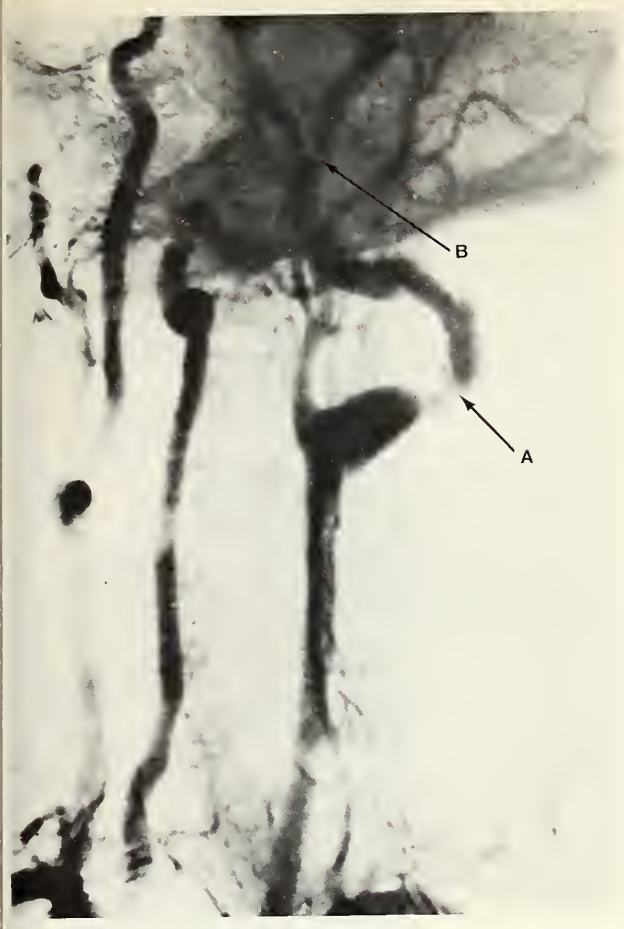


Figure 3—Preoperative arteriogram. (A) represents origin of dissection. (B) represents the reentry point.

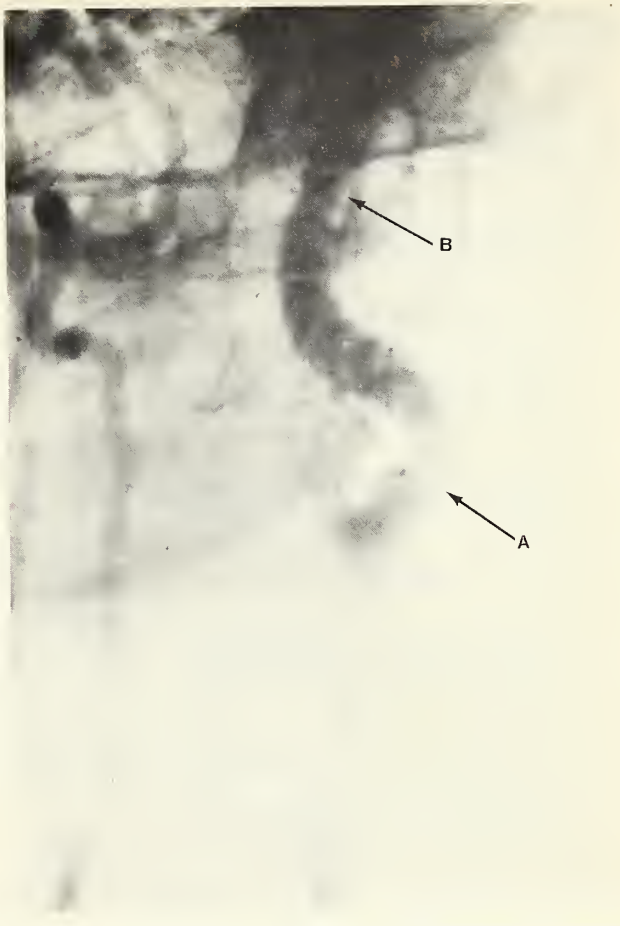


Figure 4—Postoperative arteriogram. (A) represents the internal carotid anastomotic line. (B) represents the reconstructed reentry point.

of the internal carotid artery did not occur and the lesion is accessible. Otherwise, the internal carotid artery may be ligated if the lesion cannot be reached through the cervical approach and the stump pressure is 65 mm Hg or greater.

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Acknowledgement: The authors wish to express their gratitude to Mr. Bruce Gibson for the drawings made of selective arteriography used in this publication.

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The Omental Band Sign in Reticulum Cell Sarcoma

RICHARD J. D'AMICO, M.D., Bayonne*

Ultrasound B mode imaging is an important, noninvasive technique for the visualization of intra-abdominal masses. Our patient had reticulum cell sarcoma with diffuse involvement of the omentum. Neoplastic infiltration of the omentum can be identified by gray scale ultrasonography and differentiated from other entities.

A recent article has described the omental band as a new sign of metastasis.[1] This article confirms the presence of the omental band as a sign of metastasis and differentiates it from other entities such as omental cyst or ascites. The patient to be described had reticulum cell sarcoma involving the entire intraperitoneal area, including the omentum. In this case, the ultrasonic images disclosed an omentum thickened by diffuse cellular infiltration.

CASE REPORT

A 56-year-old man was admitted to the hospital because of repeated episodes of nausea and vomiting accompanied by abdominal cramps. The abdomen was distended due to a large mass which was palpated from the level of the costal margin to below the umbilicus.

Abdominal and pelvic ultrasound were performed using Picker Corporation gray scale B mode equipment.

An anterior mass, measuring 21 cm. in diameter and irregularly shaped and marginated, was seen to correspond to the distribution of the greater omentum. The internal architecture of the band, which measured four cm in thickness (figure 1), showed nonstructured, low-level echoes. At surgery an enormous apron of solid neoplastic tissue replaced the omentum. The entire peritoneal cavity was obliterated by a layer of tumor.

Histological examination disclosed sheets of neoplastic cells infiltrating omentum (figure 2). Many of the cells were polygonal to round, with some oval to elongated. In some areas, the cells were pleomorphic; certain cells resembled mature lymphocytes, but the overall appearance was that of medium to large cells. The diagnosis was diffuse large cell lymphoma (reticulum cell sarcoma) in the omentum.

DISCUSSION

Our patient illustrates the omental band as a sign of metastasis. The thickened omental band with low-level echoes is probably a good indication of neoplastic infiltration of the greater omentum. The band signs represent a band-shaped configuration on transverse sections of a thickened omental apron.

With proper near gain suppression, low-level echoes can be visualized. These echoes in a thickened band, anteriorly and laterally, suggest the correct diagnosis.

The acoustical properties of the thickened omental band should help differentiate this entity from omental cyst, which is sonolucent, echo-free, and well-defined.[2] The internal architecture also should differentiate this entity from ascites, which has a unique distribution.[3]

SUMMARY

1. Metastatic involvement of the omentum can be detected by a non-invasive technique, namely ultrasonography.

*Dr. D'Amico is Assistant Director, Department of Radiology and Nuclear Medicine, Bayonne Hospital. He may be addressed at 50 Boulevard, Hasbrouck Heights, New Jersey 07604.



Figure 1—Transverse sonogram obtained one centimeter above the umbilicus. The right and left sides are marked (R,L). The anterior abdominal wall (aaw) and the superior aspect of the left kidney (LK) are identified. The omental band is identified and is shown to contain low-level echoes (lle). Ascites is present in the left flank region.



Figure 2—Photomicrograph of pathologic specimen showing varying sized neoplastic cells infiltrating the omentum. The overall appearance is that of medium to large cells consistent with the diagnosis of diffuse large-cell lymphoma (reticulum cell sarcoma) of the omentum.

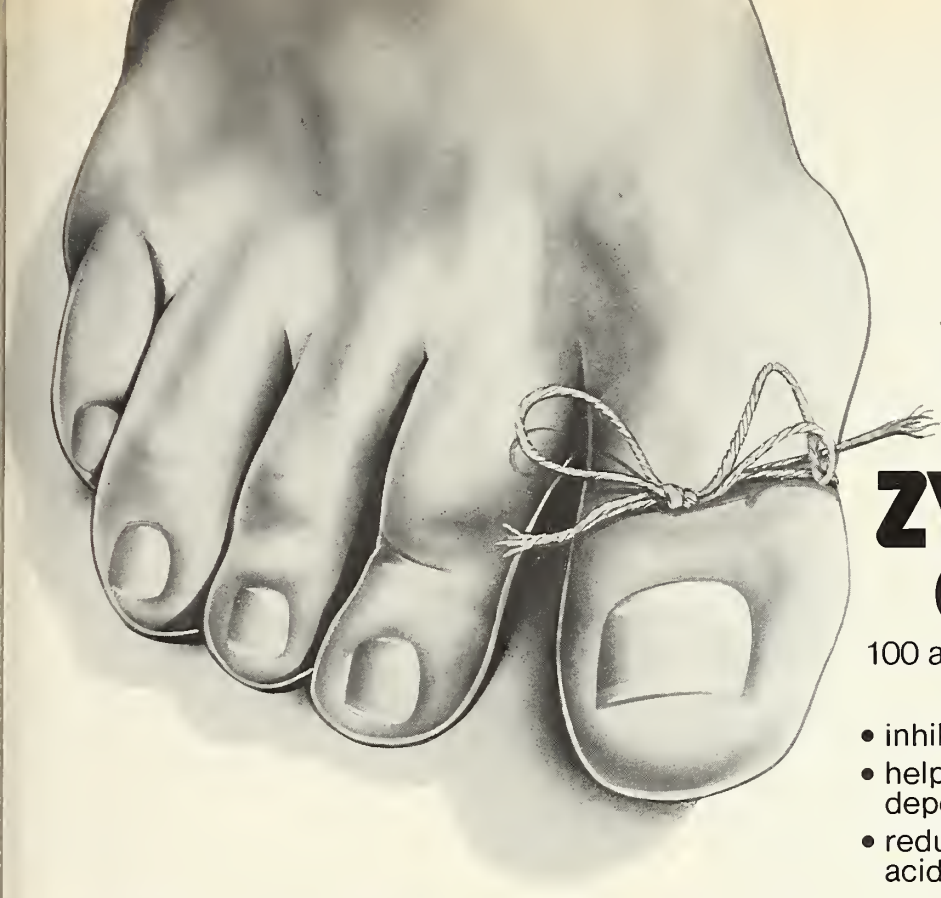
2. The presence of an omental band containing low-level echoes can help differentiate this entity from omental cyst and ascites.

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A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.
Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.
A few cases of alopecia with and without accompanying dermatitis have been reported.
In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim® (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

HOW SUPPLIED: 100 mg (white) scored tablets, bottles of 100 and 1000; 300 mg (peach) scored tablets, bottles of 30, 100 and 500. Unit dose packs for each strength also available.

Complete information available from your local B. W. Co. Representative or from Professional Services Department PML.

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Penile Metastasis from Carcinoma of the Rectum*

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BERNARD D. PINCK, M.D., Passaic

The infrequent occurrence of penile malignancy secondary to a rectal lesion prompted the present report. The carcinoma in the corpus cavernosum was derived from a tumor of the rectum and was presumably one aspect of diffuse metastatic spread.

Penile metastasis from colorectal malignancy is rare. Since the report of Abeshouse and Abeshouse in 1961, which culled from the literature 24 cases of penile metastases from gastrointestinal malignancy, few additional cases of this entity have been recorded.[1] Weitzner added four patients with large bowel metastases in his extensive review of secondary carcinoma of the penis from all types of malignancy.[2] This case presentation represents the 29th reported case of metastasis to the penis from a rectosigmoid neoplasm.

CASE REPORT

A 72-year-old male underwent a proximal diverting colostomy for inoperable carcinoma of the rectosigmoid in May 1976. His condition was stable until three weeks prior to hospitalization when he began experiencing anorexia with an associated 20-pound weight loss, increasing difficulty in bowel evacuation, and urinary incontinence.

Past medical history included bilateral inguinal herniorraphies and a cerebrovascular accident with minimal residual right hemiparesis several years previously. Physical examination revealed a thin male in no distress. There were no abdominal masses or enlarged organs. A functioning, healthy-appearing colostomy stoma was present in the right lower quadrant. The prostate was moderately enlarged, hard, and nodular. A non-tender, fixed 2 x 2 cm. nodule was palpated on the left lateral aspect of the mid-portion of the penile shaft. The nodule appeared to arise from the corpus cavernosum; the overlying skin was not fixed to it. Gross neurologic examination was normal.

Hemogram and serum chemistries were normal. Chest x-ray demonstrated nodules in both the left upper lobe and right basilar segments. Lumbosacral spine films revealed spondylolisthesis between L5 and S1 and a collapsed D12 disc space suggestive of metastatic disease. Liver scan indicated an extrinsic mass compressing the left hepatic lobe. Electrocardiogram demonstrated an old anteroseptal myocardial infarction. Intravenous urography disclosed a normal left renal unit with distal ureteral deviation and a non-functioning right kidney. A moderate residual was present on the post-voiding film. Ultrasound examination confirmed a hydronephrotic right kidney. Cystoscopy revealed detrusor hypertrophy and a nodular irregularity involving the trigonal area.

Transurethral resection of the prostate and excision of the penile nodule were performed on October 12, 1976. The lesion was found to involve Buck's fascia and the body of the left corpus cavernosum itself. Postoperative recovery was uneventful.

Microscopic examination of the penile lesion (Figure 1) revealed nests of poorly differentiated signet ring cells with mucin production dissecting a fibrous muscular stroma.

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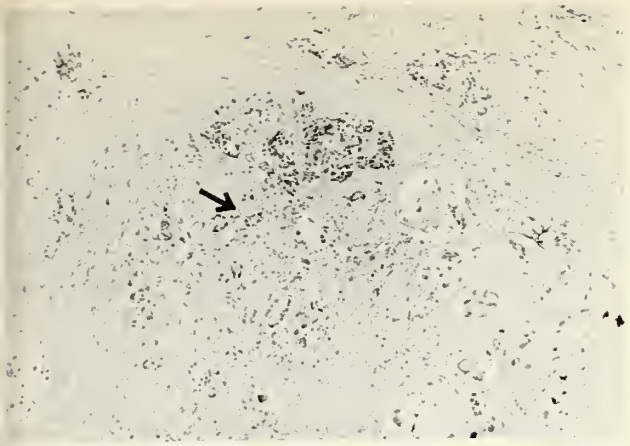


Figure 1—Nests of metastatic anaplastic adenocarcinoma in a desmoplastic fibrous stroma. Some tumor nests appear to lie in intravascular spaces.

(Figure 2) The histology was identical to that of the poorly differentiated adenocarcinoma of the rectum. (Figure 3) As an incidental finding, pathologic analysis of the resected prostate tissue disclosed a tiny focus of adenocarcinoma consistent with metastasis from the primary rectosigmoid tumor.

COMMENT

Except for several patients who have survived beyond a year following wide local excisions or amputation for a well-circumscribed metastasis, most patients presenting with secondary penile lesions have died within six months of discovery. This patient's clinical course suggests that the penile metastasis is just one aspect of widespread secondary distribution of metastatic disease.

The most common symptom of penile metastasis from visceral neoplasia is priapism, which occurs in 40 percent of cases.[2,3] Urinary outflow symptoms, when present, usually represent more proximal urinary tract involvement.

The pathways of metastasis to the penis from intestinal neoplasms have been well described.[4] The presence of a nodule in the penile corpus in conjunction with other evidence indicating diffuse carcinomatous disease implies a vascular route in this case. The pathologic study suggests a route of spread by tumor thrombosis of the cavernous vascular spaces with blood-borne metastasis.

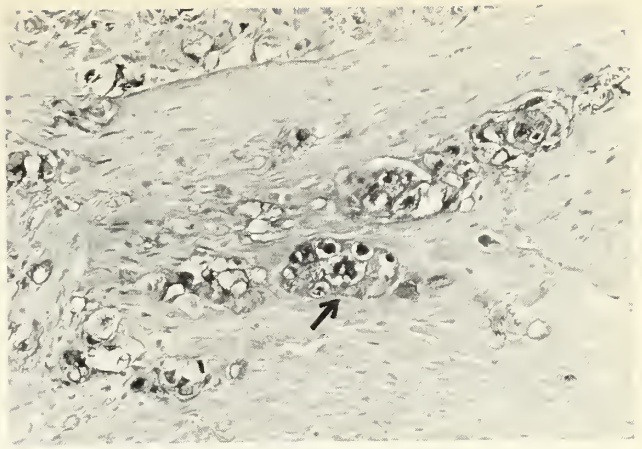


Figure 2—Metastatic mucinous adenocarcinoma. Note central acinus-lined signet cells positive for mucin.

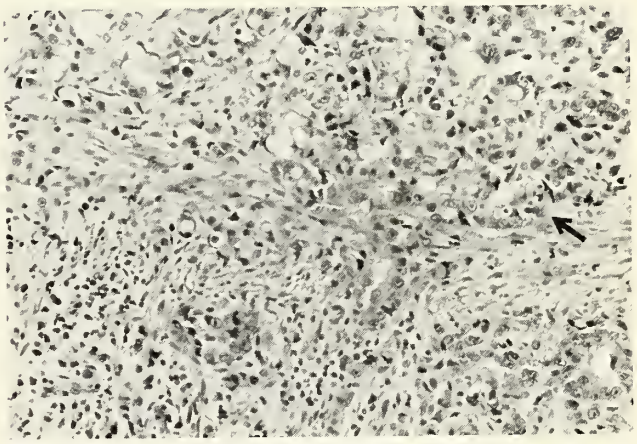


Figure 3—Primary adenocarcinoma of rectum, the original lesion. Note malignant transformation of glandular crypts in upper half of picture with invasion into submucosa.

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REVIEW ARTICLE

Chemoprophylaxis of Bacterial Infections

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Antibiotic prophylaxis is a complex subject. Evolving principles and recent data suggest several areas of medical care in which prophylaxis is effective, and others where it is valueless. Knowledge of these principles and access to the data will allow the physicians to make rational decisions. This review is intended to facilitate these goals.

The prevention of infectious disease is clearly more desirable than treatment after the fact. The discovery of antibiotics led to much optimism regarding management of infectious disease and these hopes continue to encourage physicians toward the use of antibiotics in prophylaxis of bacterial infections. These expectations have been fueled by areas of success but, until recently, the principles of effective prophylaxis have not been well defined.

In the following review, I will discuss chemoprophylaxis of bacterial infections with emphasis on principles. The areas to be explored include the "double-edged sword" of antibiotic use, the historical principles of prophylaxis, recent knowledge amending these principles, and current status of prophylaxis.

BACKGROUND

Antibiotics were introduced to clinical medicine in the late 1940s. The discovery that the extracellular products of microorganisms can influence the growth of others began a revolution in medicine. As usual, however, the reaction to the discovery was overstatement. It should have been obvious that the producing microorganisms would be protected from their own products or they would self-destruct. Therefore, these products necessarily would have a limited spectrum (i.e., organisms related to or the same as the producers would not be destroyed by the product). In addition, it was well known that the body surfaces were covered with bacteria living commensally, and occasionally demonstrating specific value (e.g., production of Vitamin K), but at first, no understanding existed of the detrimental effect of inadvertently destroying some of these bacteria by the use of antibiotics. Within a few years, however, both of these factors were noted: (1) that antibiotics do not kill all bacteria, and (2) that normal flora is damaged by antibiotics and this may be detrimental.

In 1954, Weinstein, Goldfield, and Chang[1] reviewed "infections occurring during chemotherapy" and coined the term "superinfection" to describe this event. They described the situation: "The administration of an effective antibiotic agent . . . in addition to eliminating the causative organisms produces a profound alteration in the composition of the bacterial population that normally inhabits certain tissues and organs. This byproduct of chemotherapy is frequently of no clinical consequence but on occasion is responsible for the superimposition of a serious infection on the one for which treatment was initially instituted."[1] The belief that these occurrences were rare was the subject of a study they carried out. They found a superinfection rate of two percent in all hospitalized patients treated with antibiotics but noted great variation, depending upon the site and nature of primary

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infection. They warned that the ". . . appearance of a superinfection during the administration of an antibiotic may convert a benign self-limited disease into a serious, prolonged or even fatal one."[1]

Louria and Kaminski, in 1961, assessed superinfection and demonstrated that the main determinant was the amount of antimicrobial administered rather than the illness severity or the age of the patient.[2] They noted that superinfection rates were directly proportional to the dosage of antimicrobial, its duration of use, and the broadness of its spectrum of activity. They attributed these effects as related to the degree of destruction of normal flora.

Sprunt, in 1971, noted that normal flora provided a host defense against infection, and that use of antibiotics in prophylaxis altered normal flora allowing bacterial overgrowth.[3] Prevention of overgrowth prevented superinfection. They implied that normal flora were, in some way, protective.[3] (Simplistically, it might be assumed that there are a limited number of attachment sites for intestinal or respiratory flora, and that removing some of that flora might allow new environmental organisms, resistant to the antibiotic being used, to colonize and subsequently to infect. In addition, it might be possible for resistant normal flora, present in small numbers, to overgrow during therapy and repopulate the area. This flora, although technically "normal" would now be abnormal in frequency and might lead to the development of disease.)

An almost incredible example of these principles was described in 1970 by Price and Sleigh.[4] They reported an outbreak of a Klebsiella infection in a neurosurgical unit. In 1966, prophylaxis using ampicillin and cloxacillin was begun to prevent postoperative infection in a 75-bed neurosurgical hospital near Glasglow. Klebsiella began to be isolated in 1968 and led to some fatalities. The hospital was closed and renovated, however, soon after reopening, Klebsiella again appeared. In October to May 1968-69, a large percentage of patients developed infections. Therapy for Klebsiella did not help. Prophylaxis with high-dose colistin (to which the organism was sensitive) did not help much either. Antibiotics, both prophylactic and therapeutic, were then stopped. This was "ruthlessly adhered to." There followed a remarkable reduction in all infections as well as infections due to Klebsiella. Rigid adherence continued for four months and then slightly was relaxed to include treatment for radiographically proved pneumonia, meningitis, and cerobrospinal fluid rhinorrhea. Price and Sleigh concluded that ". . . by withdrawing all antibiotics the more antibiotic-sensitive and perhaps less virulent bacteria were allowed to thrive. In turn, the changed ecological situation resulted in a reduction in the numbers of infections due to these more resistant gram-negative bacilli."[4]

In an editorial, [5] there was concurrence with the author's conclusions, but a commentary was added on the principles of prophylaxis known in 1970: prophylaxis should be used for clear and concise indications; and, the drugs should be aimed with precision at a particular organism or group of organisms. The editorial writer noted that the clinician's good intentions with regard to general prophylaxis are ". . . thwarted by the infinite variability of microorganisms and the selection of antibiotic-resistant bacteria, once the soil has been prepared by the removal of antibiotic-sensitive and often harmless commensals."[5]

In 1974, Stoddart[6] went so far as to plead for the abandonment of prophylactic therapy with broad-spectrum antibiotics, especially in intensive care units, because of the evidence that such prophylaxis may be responsible for increases in infection in such units. Petersdorf[7] had long ago indicated that routine use of prophylactic antibiotics may lead to a false sense of security with delay in diagnosis and treatment of bacterial complications. He thought that a much better policy was watchful waiting and vigorous treatment of specific intercurrent infections if they developed. He based his comments on many studies including one of his own of prophylaxis in unconscious patients in which he found more pneumonia, cutaneous infections, and bacteremia in the treated patients than in the untreated controls.[7]

Davis and Wedgewood reviewed the literature and did their own study[8,9] which demonstrated no value for antibiotic prophylaxis in viral respiratory disease.

PRINCIPLES OF THE EARLY 70s

The principles noted above became the state-of-the-art for the early 1970s. Weinstein[10] commented that prophylaxis had been used for four purposes with only the first of the four known to be successful: (1) to protect the healthy patient against invasion after exposure to a specific organism, (2) to prevent secondary bacterial infection in patients ill with viral disease, (3) to reduce the risk of infections in chronic disease, (4) to inhibit the spread of disease from a local area or to prevent infection after trauma (accidental or surgical).

Mortimer[11] noted that antibiotics used as prevention had not been as effective as therapeutic use. He stated two specific situations where prophylaxis had proven efficacy: (1) the situation of a patient exposed acutely to a specific organism and (2) the chronic or recurrent exposure to a specific organism of known and constant sensitivity. In contrast, he noted, that compromised hosts who were exposed over a period of time to multiple organisms of varying and changing sensitivities would not usually benefit from prophylaxis. Examples cited of situation (1) included transient exposure to group A streptococcus, M. tuberculosis, meningococcus, gonococcus, and others;[12,13] of situation (2) included continuing exposure of the rheumatic to group A streptococcus, of the patient with CSF fistula to pneumococcus, and of the splenectomized child to pneumococcus.[12,14] It also had been shown that attempts to prevent infection in patients with burns, acute viral respiratory disease, coma, nephrosis, and so on, were often ineffective and might do harm.[12]

NEWER MATERIAL

Since 1970, there has been much additional work (some of it earlier data rediscovered or reemphasized) which has broadened knowledge and expanded the use of prophylaxis. It has become clear, however, that the details of such use are very important. The dangers of careless antibiotic use persist and only with understanding of dynamics of antibiotic effects[15] can the narrow path between underuse and overuse be found. Wehrle,[16] in a recent commentary, stated that the overwhelming evidence for the dangers of antibiotics were ample jusitification for limiting chemoprophylaxis to circumstances where justifiable use has been *documented.*

In studies done during the 1950s, Burke[17,18] noted that despite the fact that antibiotics prevent bacterial growth and that logic would lead one to use them to prevent bacterial complications, they have proved ineffective. He stated that, from the biology of tissue defense, the reasons for this failure are "obscure." He looked to the question: ". . . is there any time during the period of development of a primary bacterial

lesion in which systemically administered antibiotics are effective in preventing or inhibiting the development of the lesion?"[17] In his study of experimental lesions, he noted a short period of time (the decisive or critical period) in which there was intense tissue defensive activity. He was able to show that the ultimate size of a lesion was the result of the effectiveness of this defense. Events during this period were at a molecular level and often had little immediate effect on the morphology of the area. The period was over long before classic histologic evidence of inflammation was demonstrable. Burke concluded that events occurring before and for several hours after an experimental bacterial wound infection affected the lesion size at its maturity (24 hours) and that events occurring after three hours postinfection had no influence. Further, he demonstrated: ". . . that the moment of contamination is the time when the tissue defensive mechanisms, if functioning, have the highest potential of preventing the development of a bacterial lesion . . .".[17] This moment of contamination and the three hours after were called the effective period of preventive antibiotic action. Burke concluded with a statement of principles for surgical prophylaxis.[18] This noted that the natural host resistance was important and reduced by anesthesia and surgery. Infection risk could be reduced by supplementing resistance if the supplement was delivered before contamination occurred. Using such prophylaxis was reasonable only if the risk of infection exceeded the risk of damage from the antibiotic. He concluded that "preventive antibiotics are indicated if there is a high probability that a patient's natural resistance to bacterial invasion will not overcome the combined bacterial and physiologic challenge of a surgical procedure."[18] He indicated three categories of surgical patients who might be candidates for appropriate prophylaxis: patients with compromised defenses; patients whose surgery may lead to massive contamination; patients who will have extensive surgery.

An extension of Burke's principles specifically related to surgery has been summarized recently by Clarke.[19] Since the bowel normally is populated by large numbers of bacteria which usually are excluded from the body by the endothelium, disruption of the endothelium might lead to invasion by these bacteria. A reduction in the numbers of such bacteria at the time of surgery might reduce the incidence of invasion.[19] (Timing is critical. The use of preparations too early might reduce the numbers of bacteria only to be promptly followed by growth of resistant flora, both endogenous and environmental. Bowel preparation done too late might not influence the numbers of bacteria. And, continuation of antibiotics too long after preparation might encourage superinfection by mechanisms previously discussed.)

These principles of operative prophylaxis have been tested in a variety of circumstances and probably have applicability to other medical and surgical situations.

Today, the facts regarding prophylaxis for a single species in a transient exposure situation remain undisputed: These include acute exposure of normal hosts to group A streptococcus, meningococcus, Mycobacterium tuberculosis, gonococcus, typhus, syphilis, diphtheria, and pertussis.[20,21] The benefit of prophylaxis is currently unclear for H. influenzae type b, Shigella, Staphylococcus, trachoma, and Clostridia.[20] Prevention of infection caused by a limited number of species of organisms in some compromised hosts also may be effective: This includes group A streptococcus (in rheumatics, burns), pneumococcus (in CSF rhinorrhea, sickle cell anemia, splenectomy), strep viridans (in congenital and rheumatic heart disease), E. coli (in repeated urinary tract infections, enteric infections) and, perhaps, Staphylococcus aureus (in neonates).[20-25]

In the compromised host at continuous risk for a multitude of organisms the data are conflicting. (The principles noted above would suggest that continuous prophylaxis would lead to overgrowth of resistant organisms and an increasing risk of infections with such agents. But, there may be circumstances where antibiotics may act as antiseptics because concentrations reached effectively may sterilize the area, e.g., the bladder. Under such circumstances repopulation with resistant flora would be unlikely to occur.) The details of the prophylaxis appear to be most important.

In the following list of some recent indications, specific references should be sought to define the nature of such prophylaxis. Effectiveness has been demonstrated in: granulocytopenic febrile patients,[26] granulocytopenic patients with acute myelogenous leukemia[27] (but not in undifferentiated acute leukemia).[28] Foley catheterization (short term only),[29] recurrent otitis media,[30] recurrent urinary tract infections,[31] endotracheal intubation[32] and tracheostomy (short term only).[33] In the newborn, evidence for value has *not* been demonstrated for umbilical arterial or venous catheterization,[34-36] or for premature rupture of the membranes, group B streptococcal infections, prematurity, respiratory distress syndrome, and exchange transfusion.[37]

It is in surgery that much new data are available which suggest the value of prophylaxis heretofore doubted. Burke's principles for prophylaxis usually have been demonstrated correct. Patients with compromised defenses, massive contamination and/or extensive surgery appear to benefit from specific prophylaxis appropriately timed.[15] Those not at risk do not appear to be helped. Evidence for value in surgery has been shown in compromised hosts: in renal transplantation (wound infection reduced),[38] in gastric surgery (patients with decreased acidity),[39] and in cesarean section;[40] in massive contamination:[41] in colon surgery,[19,42] in cholecystitis,[43] in vaginal hysterectomy,[44] and in pulmonary resection;[45] in extensive surgery: in open heart surgery,[46] in pharyngeal and laryngeal surgery,[47] in exploratory laparotomy,[48] and in orthopedic procedures.[49] Prophylaxis has not been demonstrated to be of value in abdominal hysterectomy,[50] closed heart surgery,[51] and cardiac catheterization.[52] Surprisingly, some value has been shown in situations not clearly in the above categories, e.g., appendectomy.[53,54] (The explanation here may be a combination of two principles: (1) although massive contamination does not occur, some peritoneal contamination is likely, and this may be reduced by prophylaxis; and (2) these patients are usually healthy and the risk of superinfection is, therefore, small.) Antibiotics, on balance, may be effective.[55] However, consistent with Burke's critical or decisive period is a study by Samson[56] which demonstrates no value for antibiotic prophylaxis of minor lacerations. This confirms earlier work by Day.[57]

It cannot be overemphasized that careful selection of drugs effective for organisms expected to be found in the anatomic location[58] is crucial, and as noted above, the time of initiation and discontinuation of the prophylactic agent is critical. In a recent study, Stone [59] prospectively evaluated 400 patients who were randomized to receive prophylaxis given 12 hours preoperatively, just prior to operation, after operation, or not at all. The 400 patients had varied operative procedures. The results were a reduction in infection in the preoperative and intraoperative antibiotic group.

CONCLUSIONS (see Table I)

Conclusions for surgical prophylaxis drawn by Ledger[60] remain valid. His guidelines are similar to Burke's. He would attempt prophylaxis if the operation carried significant risk of postoperative infection or if it caused significant bacterial contamination. He stated that the antibiotic used should be effective for the expected contaminating organisms, it should be present at the time of incision, and it should be used for a short term.

Unfortunately a recent report by the British Study Group on the use of antimicrobial drugs,[61] showed that antimicrobial prophylaxis was used in random fashion in five London teaching hospitals. "Striking differences were found, particularly in the duration of treatment in different hospitals in the same specialty, and also . . . in the same hospital." The conclusion of the study group was that the reasons for use in some of these hospitals were that doctors believe that antibiotics destroy bacteria and, therefore, must help. Doctors often oppose clinical trials because they want their patient to have the best. The study group felt that it was only by suitably controlled clinical trials that answers could be obtained.[61]

SUMMARY

Chemoprophylaxis of bacterial infections has undergone significant change in the last decade. The effective and appropriate use of antibiotics to prevent infection has been expanded to include surgical indications, and prevention of infection in compromised hosts. Principles of prophylaxis have been identified and the details regarding the nature of the antibiotic and the duration of its use are being worked out. Controlled prospective trials to define specific indications and the details of use are vital.

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Table I
*Chemoprophylaxis of Bacteriol Infections**
(1978)

I. Tronsient exposure of normol hosts to specific agents
 A. Strong evidence
 1. group A streptococcus
 2. meningococcus
 3. M. tuberculosis
 4. gonococcus
 5. ogent of typhus
 6. agent of syphilis
 7. C. diphtheriae
 8. B. pertussis
 B. Equivocol evidence
 1. H. influenzae type b
 2. Shigella
 3. Staphylococcus
 4. C. trochomotis
 5. Clostridia
 6. E. Coli

II. Tronsient exposure of compromised hosts to specific agents
 A. Strong evidence
 1. group A streptococcus (burns, rheumatics)
 2. pneumococcus (CSF rhinorrheo, sickle cell disease, osplenio)
 3. Strep viridans (heort disease)
 4. E. coli (repeot UTIs)
 5. S. oureus (neonates)
 B. Evidence not supportive
 1. group B Strep (neonates)

III. Continuous exposure of compromised host to multitude of ogents**
 A. Preponderance of evidence fovors use
 1. febrile granulocytopenic potients
 2. acute myelogenous leukemia and granulocytopenia
 3. Foley cotheters (short term only)
 4. recurrent otitis medio
 5. recurrent urinory infections
 6. endotrocheal intubotion ond trocheostomy (short term only)
 B. Preponderonce of evidence not supportive for prophylaxis
 1. umbilical ortery or vein cotheterization
 2. premoture rupture of membranes
 3. prematurity
 4. respiratory distress syndrome
 5. exchonge transfusion
 6. undifferentioted acute leukemio ond granulocytopenio

IV. Surgicol prophylaxis***
 A. Compromised defenses
 1. renol transplontation
 2. gastric surgery (potients with decreosed acidity)
 3. coeserian section
 B. Massive contominotion
 1. colon surgery
 2. cholecystectomy
 3. voginol hysterectomy
 4. pulmonary resection
 C. Extensive surgery
 1. open heort
 2. phoryngeol ond laryngeal
 3. explorotory loporotomy
 4. orthopedic procedures
 D. None of the obove categories
 1. evidence fovors
 a. appendectomy
 2. evidence ogoinst
 o. obdominol hysterectomy
 b. closed heort surgery
 c. cardioc catheterizotion
 d. minor lacerotions

*detoils of cirmcumstonces for or methods of prophyloxis are not included here.
**details of methods ore criticol. See originol sources.
***detoils of specific indications ore essential. Consult references.

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Farewell Address*

Charles S. Krueger, M.D.
President 1978-1979

The 213th year is past and the problems and events have been such that the Medical Society of New Jersey has responded in a way in which all members can be proud. Ours is the single organization truly representing all physicians in New Jersey. We are over 9,000 strong. Our opinions are sought on all medical matters coming before the public. Your leadership has responded positively and aggressively, addressing the problems which we feel will affect the better health of our patients and the public at large. It is of utmost importance that our general membership know how we have acted and it is through this House of Delegates that I hope we can provide this information and exert positive leadership.

Our new home in Lawrenceville is now a reality. Though yet not completed, enough has been done so that we all can be proud of our new facility. It is not only a good investment, but will allow us to support functions "at home," long overdue. We have the capacity to hold the House of Delegates in our headquarters building, so we now should give strong consideration for a fall-winter interim session.

PROFESSIONAL LIABILITY

Professional liability continues to remain our most active area and one that is close to all of us. We can be proud of our insurance company, the Medical Inter-Insurance Exchange of New Jersey and the New Jersey State Underwriters, Inc. Your insurance company continues to prosper, and for the first time in many years, perhaps ever, there has been a reduction in insurance premiums. Some of the high-risk groups and, by class transference, one of the lower-risk groups (the pediatricians) have no change in premiums, but in most specialties, physicians will find an overall eight percent reduction The management of our company is excellent and the efficiency in handling claims has been nothing short of unbelievable. To have such cooperation with an insurance company in such a sticky area is reassuring. Just ask anyone who has had a recent claim against him and you will be very proud of your company. We hope by increased participation in risk management and in physician education that our malpractice experience will decrease and that the premiums will be reduced further. To the contrary, the New Jersey State-run insurance company is having difficulty keeping a carrier to insure their physician clients. Presently there is coverage with a reduced premium, but an additional billing is anticipated later in the year.

The Supreme Court pre-trial screening panels to precede medical malpractice court action finally have become a reality. We have panels consisting of an active judge, a practicing lawyer, and a practicing physician that already have made decisions in selected cases, which seem to be favorable to the medical profession. It is hoped that a fair hearing will take place without the emotions generated in the courtroom so that those with true merit of malpractice will be settled quickly to the benefit of the patient, and those without merit dismissed.

The malpractice legislation is still slow in coming, but two bills have been introduced and a third is being prepared for introduction. The two bills that have been introduced would give immunity to committee members conducting peer review and would allow physician defendants to recapture costs of defending frivolous suits. The third bill to be introduced would produce a statute of limitations of between three to five years.

GOVERNMENT IN MEDICINE

The single most important area now confronting us is the increasing involvement of government in the private practice of medical care. It is understandable because the dollars spent on medical care are increasing at a rate which is now over eight percent of the national budget. Inflation has made the public sit back and take notice and now, perhaps for the first time, there is a general awareness that there is a limit on what we can spend and on the amount we wish to be taxed. Americans want the best medical care in the world but now realize that this comes at a price which is dear to all of us. We are willing to spend more on health but, in order to do this there must be sacrifice. If the government is going to continue to increase its spending, it is logical that taxes must be increased in order to support this. Many Americans realize that less seems to be returned from Washington compared to what goes down. It is in this atmosphere that we are finding ourselves almost in an adversary position with government, and it is in this atmosphere that discussion about national health insurance is taking place.

I had the opportunity this past year to observe firsthand the National Health Service in Britain and the National Health System in Canada. Their doctors generally have fared well. Of course, no one is getting rich, but the impression is one of moderate content with their systems. Both countries have social systems that offer additional compensations, so that dollar-for-dollar income comparison is difficult to perceive. There is little professional satisfaction in their medical practice, and an outsider is struck immediately by their low productivity. In addition, they work fewer hours and, therefore, patients are forced into long lines, and thus the long wait for elective medical care and surgery. The British emergency system is excellent, but this is where it ends. There is no longer pride in medical practice and the private initiative in individual medical services has disappeared.

The American public is running scared. They are frightened of the rising cost of medical care and possibly their inability to afford it. Catastrophic illness is just what the word implies, but the fear is not of medical catastrophe, but of financial catastrophe. We must offer answers. We must agree to work on some type of health-care coverage which responds to this most pressing problem.

*Presented before the House of Delegates, May 13, 1979, Atlantic City.

We must convince the public that we have their concerns uppermost in our minds and that, when in a public forum, we don't appear to be self-serving. For some time, I have felt that a doctor who is satisfied, who has taken great pride in his services, who is compassionate, and, yes, financially rewarded, is much better in offering his services to patients. Any system which tries to take away this freedom quickly becomes inferior, and in the end would be completely unacceptable in this country.

Our government leaders, I believe, are well aware of this. The leadership of your Society has met numerous times this past year with our government representatives in this State, as well as in Washington. I personally had several discussions with Representative Paul Rogers, and even spent an evening with one of our greatest adversaries, Joseph Califano. I believe we have had some degree of influence on policies which refer to hospital cost-containment as well as certificate of need.

A government-controlled health care system as proposed by Senator Kennedy and others is completely unacceptable to us. We agree that there are a significant number of people without adequate health insurance coverage in this country, but this is not to say that our whole system must be disrupted to include these people in a total coverage system. Alternatives must be offered to cover this significant minority, however, and we must come up with ways to correct this injustice, while preserving the best parts of our present system.

In January, your Board of Trustees appointed an ad hoc committee to develop a position paper on national health insurance to present to the AMA for their presentation to Congress. It contains four important points:

1. Minimum standards of adequate health benefits should be required of all insurance policies sold in the United States, with appropriate deductible and coinsurance.

2. A simple system of uniform benefits should be provided by government for those unable to provide for themselves.

3. Catastrophic insurance through private carriers, with government supplement when necessary, should be standard for all.

4. Administration of the above programs should be at the state level.

Because financial awareness and incentives are becoming so important, we must look to alternative health care delivery systems. New Jersey is second only to California in the number of HMOs, the prepaid health care system developing in our country. Most of us are uncomfortable with these closed-panel systems, but the open-panel HMOs or independent practice associations (IPAs) may offer the best of both worlds. I am concerned that physicians are being blamed for the increase in medical cost while receiving less and less of the total health care dollar. It has been shown that HMOs might reduce the total cost of health care. The free market system competing for the health care dollar is by far more attractive to me and I believe acceptable to the American public, compared with a government handout with all its controls and regulations, and increased taxes.

NEW JERSEY FOUNDATION AND LEGISLATION

Our New Jersey Foundation for Health Care Evaluation presently is exploring the feasibility of a statewide fee-for-service HMO, contrasting with regional county(ies) IPAs. It appears attractive to me and presents a full-care system to the public which contains the free choice and quality of our present system, along with the financial incentives and cost-containment options so necessary at this time. I hope we will study it carefully and participate if it proves viable. The Foundation needs our full support to develop it.

Two of our biggest problems this past year revolved around the new Public Law 94-484, which severely restricts the number of foreign-born, foreign medical graduates entering the mainstream of medical practice in this country. The New Jersey Department of Higher Education and others see this as a real threat to the physician supply in our State. There is ample support for this because recently the number of FMGs has amounted to over 70 percent of the newly licensed physicians, with only 10 percent of these being American born. We also have the dubious distinction of having the greatest relative number of FMGs in our residency programs. This, of course, has not been all that bad. However, in future years, it is estimated that because of the restrictive nature of PL 94-484 our new physician supply will be curtailed severely. Because of this, there has been considerable impetus, through proposed legislation, to allow the physician's assistant to practice in this State.

It appears we have won the first battle opposing physicians' assistant legislation in New Jersey. This past Fall, after considerable study and discussion, I personally had felt that we had no chance of defeating this legislation and that our best form of attack was to give the legislation conditional approval, requesting additional restrictive amendments. These amendments were accepted. It became increasingly apparent, however, that this House of Delegates had great concern about physicians' assistants and that our Society should continue active opposition. This House stated the quality of medical care would suffer, there would be no reduction in the cost of medical care, and there was no demonstrated need. It also was felt that even though the proposed legislation is very restrictive, it would be a "foot in the door," allowing future independent practice and thus creating another problem with which we would have to come to grips.

The Statewide Committee to Seek Support to Opposition of Physicians' Assistant Legislation, made up of presidents and presidents-elect of the five largest county medical societies, must be given high marks, along with the keyman system, because they were able to influence enough legislators to vote in the negative when the physicians' assistant legislation initially was brought to the floor on February 26. It was through their gallant effort, along with our excellent lobbying system, that we were able to defeat the initial vote on this bill. Another vote, however, is forthcoming, and we must not sit back and rest on our laurels, for even though the initial battle has been won, the war is not yet over.

MEDICAL EDUCATION

It appears that the education of medical and osteopathic students in southern New Jersey has been settled. The full Board of the Department of Higher Education, on April 20, 1979, approved a compromise plan agreed upon by both the staff of the Department of Higher Education and the College of Medicine and Dentistry of New Jersey.

This plan will continue all the basic science education—that is the first two years of medical school—at the New Jersey Medical School in Newark and the Rutgers Medical School in Piscataway. The osteopathic students will train in Piscataway. The medical students in the southern extension of Rutgers Medical School will take their first two years in either Newark or Piscataway, and their final two clinical years at Cooper Medical Center in Camden.

The New Jersey School of Osteopathic Medicine will be allowed to enlarge so that eventually 56 students will be graduating yearly by 1983. The entire output of physicians in this State, both M.D.s and D.O.s, will be 382 by 1983. An eight to nine million dollar building will be built to house the clinical teaching facility adjacent to Cooper Medical Center and the proposed Veterans Administration Hospital in Camden. This facility will house both the M.D. and D.O. faculty offices, small conference rooms, and the library, and will be under the supervision of the Dean of the New Jersey School of Osteopathic Medicine.

The entire southern New Jersey medical education program will come up for review by a panel of experts to be appointed by the College of Medicine and Dentistry of New Jersey, during the school year 1983-84, with special emphasis to be given to the possible need for a basic science facility in southern New Jersey.

OTHER EVENTS

Many other events have transpired this past year. Our Governor threatened the Medical Society with a lawsuit, charging restraint of trade when we proposed to *not* bill for our services to Medicaid patients, thereby causing our State not to qualify for federal support. Partly because of this and the difficulty in implementation, the Board of Trustees chose not to carry out the intent of the resolution of the 1978 House.

Our Medicaid Committee continues to be very active, meeting monthly with representatives of the Division of Medical Assistance and Health Services, which has control over Medicaid matters. A fair reimbursement fee is still the major problem and one with which we are not satisfied. We now are receiving less than 50 percent of our customary fee, and for a family practitioner this barely covers the overhead. Because of this Committee's activity, progress is being made regarding the differential in the fee for a patient's visit to a hospital clinic and to a physician's office.

We have had our problems with the Department of Health which seems to be becoming more and more involved in the private practice of medical care. It has proposed an excellent system regionalizing emergency medical services, which, in some areas, has been something long needed in this State. It has, however, overlooked the voluntary system now in operation quite successfully in many areas, and the voluntary ambulance people feel they have been bypassed in this new system. Input from our Society has not been utilized fully, and we are concerned about implementation of the new system. Also, the cost is considerable. At a recent meeting of the Health Care Administration Board, it was decided not to implement this new program at this time.

The certificate of need for large hospital expenditures seems to be working properly in most regions. We have questioned its application to private physician offices. The Medical Society of New Jersey and the New Jersey Hospital Association have joined in a voluntary cost-containment committee to address the cost of medical care as it relates to hospital practices.

For a number of years, our membership has remained stable, in spite of increasing dues. It has been reassuring to find that we have lost only a very small percentage of members because the continuing medical education requirements were not met. It should be noted that the State Board of Medical Examiners has chosen not to make this a requirement for state licensure because of our action.

We seem to be approaching a time when there is increasing competition between professional societies for membership. County and state society membership for years has almost been automatic and, therefore, we have not had to go out and seek new members. We now are seeing, however, that because of increasing dues and other commitments, physicians are starting to be selective, and in some cases this selection includes opting out of the general medical societies —that is, the county, state and national.

SUMMARY

As can be seen from this rather lengthy report, the Medical Society of New Jersey continues to be in the forefront of medical matters in this State. Our staff has enlarged. All problems submitted are considered and action generated as seems appropriate. Solutions cannot be acceptable to everyone, but, nevertheless, everyone's position is considered carefully. Yes, the squeaky wheel still gets the most attention, and our Society, through its House of Delegates and Board, being democratic, finds the wheels of decision and action at times grinding slowly but, nevertheless, continuously with positive results.

It is inevitable that our membership will not be all inclusive as it has been in recent years. However, by creating a positive, participating membership, perhaps a smaller one, our Society will be more unified and stronger. It takes money to run our organization, and presently our budget is one and one-half million dollars. If our membership decreases, our dues must increase. It is a price the membership must reckon with.

The time has come when each of us must encourage our colleagues to continue membership in the Medical Society of New Jersey, along with our county organizations. Additional consideration must be given to our national organization, the American Medical Association. Too many nonmembers are receiving benefits from our groups, and I feel that many of us do not realize how effective our groups are and how they benefit the entire medical profession. Further numbers and monies will be needed for us to remain effective.

As I said one year ago in my introductory remarks, join, participate, educate yourself in Medical Society matters— you will be rewarded by your activities.

THERAPEUTIC DRUG INFORMATION

This information is compiled by the Schwartz Inter-National Pharmaceutic and Therapeutic Drug Information Center of the Brooklyn College of Pharmacy, Long Island University.*

1. Do you have any information concerning the interaction between digoxin and spironolactone?

Spironolactone (Aldactone® and contained in Aldactazide®) has been shown to inhibit the tubular secretion of digoxin and thereby its renal clearance. The clinical importance of this interaction is not known.

Steiness[1] conducted a study to evaluate the mechanism of renal handling of digoxin and the effects of spironolactone on same. Simultaneous measurements of inulin and digoxin clearances were performed in 13 digitalized patients with chronic congestive heart disease. Nine of the patients subsequently were treated with 100 mg spironolactone daily for ten days, and the measurements of inulin and digoxin clearances were repeated. The mean concentration of plasma digoxin increased from 0.8 ng/ml to 1.0 ng/ml ($P<0.01$). The data suggest that spironolactone decreases tubular secretion of digoxin in the distal segment of the renal tubulus.

Waldorff *et al*[2] studied the effect of spironolactone on the kinetics of digoxin in eight subjects (four patients with arteriosclerotic heart disease plus four healthy persons). All subjects were given 0.75 mg digoxin intravenously. After five days of treatment with spironolactone 100 mg twice daily, the same intravenous dose of digoxin was given again during continued administration of spironolactone. The authors found that plasma digoxin levels increased by one-third during spironolactone treatment.

In conclusion, although the precise mechanism of interaction between digoxin and spironolactone is not known, there is some evidence that the combination resulted in an elevation of plasma digoxin level. Further investigations are needed to determine the clinical significance of this interaction, and it is advisable to monitor digoxin levels when adding spironolactone to the therapeutic regimen of patients on digoxin.

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2. Recently the lay press suggested that cimetidine had induced cardiac arrhythmia. Do you have any information relating to this possible adverse reaction?

Cimetidine (Tagamet® SK & F) is a histamine (H_2) receptor antagonist indicated in the treatment of duodenal ulcer and pathological hypersecretory conditions.[1,2] There is no mention of cardiovascular side effects in the official literature of cimetidine, although H_2 receptors have been documented in the heart and these can be blocked selectively blocked by H_2 receptor antagonists as shown in the isolated guinea pig atrium.[3,4,5]

Reading *et al*[6] have reported a case of bradycardia in a 49-year-old male without apparent heart disease and a normal electrocardiogram (EKG) treated with cimetidine for gastrointestinal bleeding. After ten days of therapy his heart rate was 57 per minute. On the next day, an EKG indicated atrioventricular dissociation with idioventricular rhythm with a heart rate of 50 per minute. Cimetidine was discontinued and tincture of belladonna was administered. The EKG two days later was normal and the heart rate returned to 63 per minute two weeks later.

Jefferys and Vale[7] reported bradycardia associated with cimetidine in two patients. The first was a 39-year-old male treated for gastric ulcer with cimetidine. After the second day of therapy, he developed malaise and dizziness which persisted during the four weeks of therapy. Examination revealed a pulse rate of 48 per minute (previously 76 per minute) and sinus bradycardia confirmed on EKG. Symptoms disappeared when cimetidine was discontinued and the patient's pulse rate rose to 72 per minute. In the second patient, a 62-year-old male with melena secondary to aspirin, cimetidine was administered intravenously and sinus bradycardia (pulse rate of 42 per minute) was noted four hours later. The drug was given orally during the next three days and bradycardia persisted. After the drug was discontinued, the patient's pulse rate rose to 64 per minute. Three days later, the patient was challenged with an intravenous bolus of cimetidine and his heart rate fell over the next three hours

*The Center serves as a source of intelligence on therapeutic and pharmaceutic information not readily available to physicians, at no charge to them, and provides this information with minimal time involvement. It is staffed by trained pharmacists; Jack M. Rosenberg, Pharm. D., Associate Professor and Chairman, Division of Clinical Pharmacy, Brooklyn College of Pharmacy, is Director and Walter Modell, M.D., Emeritus Professor of Pharmacology at Cornell University Medical College, is pharmacologist consultant. The service is available Monday through Friday from 9 a.m. to 5 p.m.—telephone (212) 622-8989 or 330-2735. Responses to these questions were prepared by J. M. Rosenberg, M.S., Pharm. D.; P. Sangkachand, M.S., R. Ph.; T. H. Chin, Pharm. D.; R. J. Fuentes, R. Ph.; D. L. Menkes, R. Ph.; Mollie Fisch.

from 70 to 44 per minute and returned to normal over the next ten hours.

A case of bradycardia associated with cimetidine was reported in a 33-year-old woman with kidney failure.[5] On admission to the hospital, the patient's EKG was normal and subsequent gastroscopy revealed the presence of a gastric ulcer. The patient was placed on oral cimetidine and after two weeks of therapy complained of palpitations. The EKG showed sinus bradycardia with the rate varied from 40 to 60 per minute. In spite of kidney failure, the patient exhibited hypokalemia and was treated with 60 mEq of potassium chloride by intravenous infusion. The patient later died as a result of ventricular fibrillation. The authors did not provide formal evidence of a correlation between cimetidine and arrhythmia but a relationship nevertheless appeared probable to them.

In conclusion, several reports of bradycardia have been associated with cimetidine therapy which may be related to inhibition of H_2 receptor sites in the heart. More data are required to assess the effects of cimetidine on the heart.

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3. Can you please provide any information on the effect of nitrofurantoin on DNA?

Nitrofurantoin (Macrodantin®, Furadantin®), a nitrofuran derivative, is used extensively against many strains of common urinary tract pathogens.[1] It has been suggested that most nitrofurans are mutagenic and carcinogenic, and nitrofurantoin has been shown to cause DNA breakage of human cells.

Olive and McCiella[2] have demonstrated that enzymatic reduction of the nitro group, on nitrofuran compounds, is essential before mutations or DNA single-strand breaks are induced; while the furan analogues, that they have tested without the nitro group, are neither mutagenic nor carcinogenic. They found the extent of DNA breakage decreased progressively as the oxygen concentration of the incubation medium was increased.

Hirsch-Kauffman M, *et al*[3] observed that nitrofurantoin can damage DNA of bacterial and human cells. In a bacterial model, DNA synthesis was affected more severely by nitrofuran in cells defective in DNA repair. The damage resembled that of ultraviolet (U.V.) irradiation, as both nitrofuran and U.V. irradiation inhibited DNA replication and were repairable. In human cultured diploid fibroplasts the dose of nitrofurantoin required to inhibit DNA replication was considerably higher than the dose to produce the same effect in the bacteria. This suggests that in healthy cells, the nitrofurantoin effect may be counteracted sufficiently by the repair enzymes, especially at the lower drug concentration range.

Since nitrofurantoin has been shown to damage DNA synthesis, particular care should be exercised in using this drug in patients with any DNA repair deficiency. More data are needed to determine the long-term clinical effects of these observations.

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4. What is the current status of isoprinosine for viral infections?

Isoprinosine (Newport Pharmaceuticals and Norwich Pharmacal), also known as inosiplex and methisoprinol, is an investigational drug in the United States and currently marketed on a limited basis in 31 foreign countries.[1,2]

Isoprinosine appears to potentiate cellular-mediated-immunity and is classified as an immunopotentiator.[3] The drug also may enhance host messenger ribonucleic acids for host ribosomes and thus significantly reduce virus directed protein synthesis. This effect appears to be opposite to cortisone, a well-known immunodepressant.[4]

Theoretically, isoprinosine's action should afford a broad spectrum of activity against most viruses. Numerous types of influenzas, herpes, rhinovirus, and other diseases with possible viral etiology have been tested, but with equivocal results.[2,5,6,7] The drug may not be effective as a prophylaxis agent just prior to viral infections, and indeed, may interfere with the normal host response.[8]

To date no serious side effects have been reported other than hyperuricemia.[4] Proof of efficacy remains difficult because *in vitro* models are unreliable predictive indicators of *in vivo* efficacy. *In vivo* models are also difficult to design due to individual variables such as dosing, infection, exposure time, and the patients baseline cellular immune status. Despite these difficulties, isoprinosine has been approved most recently for the treatment of measles in West Germany.

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5. Do you have any information on the use of guar gum in the treatment of diabetes?

Guar gum is a gel-forming, non-absorbable carbohydrate of vegetable origin widely used as a suspending, thickening and stabilizing agent in foods and emulsions. It has been

suggested that guar gum may be useful in the treatment of diabetes mellitus. When added to a carbohydrate test meal, it has been shown markedly to decrease postprandial hyperglycemia.[2,3] Since diabetes mellitus is uncommon in non-industrial societies, it has been theorized that this may be related to the high content of unabsorbable plant polysaccharides in their diets.[3]

Jenkins *et al*[1] studied the effect of guar gum on 24-hour urinary glucose excretion in seven diabetics. The investigation was divided into "home-based" and "metabolic ward" phases. The results from both phases indicated that the addition of 25 g. of guar gum to the diet produced a marked reduction in mean urinary glucose output. The treatment was well tolerated by most patients with flatulence being the main problem in some.

The potential mechanism of action of guar gum may be due to its effect on glucose absorption rates, release of gastrointestinal hormones affecting insulin secretion or modifying glucose metabolism, or the release of insulin.[2]

In conclusion, the addition of an unabsorbable carbohydrate (such as guar gum) to the diet may prove to be a useful adjunct in the treatment of diabetes. A reduction in postprandial hyperglycemia and urinary glucose excretion may lead to decreased insulin requirements.

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6. Do you have information about the non-steroidal, anti-inflammatory agents for dysmenorrhea?

Primary dysmenorrhea is the most common gynecological complaint, and the greatest single cause among young women of lost time from work and school.[1] The etiology is unclear, but recent studies suggested that vigorous activation of the prostaglandin generating system causes excessive myometrial contractility leading to uterine ischemia and pain. This hypothesis has led to clinical trials of non-steroidal anti-inflammtory drugs, such as indomethacin (Indocin®) and naproxen (Naprosyn®) which inhibit prostaglandin synthetase, and the fenamates, such as mefenamic acid (Ponstel®), which have an antagonistic action on prostaglandin receptors as well.[2]

Many patients with dysmenorrhea respond to aspirin by itself, or in combination with other drugs.[3] The action of aspirin as a prostaglandin synthetase inhibitor is well documented.[4]

Hill,[5] in 1973, reported that he had successfully treated several dysmenorrheic patients with indomethacin. In a double-blind crossover study, indomethacin was tested in 47 female patients, using aspirin and placebo as control drugs.[6] Aspirin was found better than placebo, but indomethacin proved to be superior to aspirin.

Henzl[7], in two independent, double-blind trials, evaluated naproxen versus placebo in 43 dysmenorrheic women, during four menstrual cycles. The patients were instructed to take initially 550 mg of the drug, followed by 275 mg every six hours, for a maximum of five days. According to the scale utilized to rate pain intensity and menstrual cramps, naproxen was significantly better than placebo in both studies. Another trial of naproxen versus placebo was conducted in 17 patients with dysmenorrhea.[8] The mean relief score for the naproxen group was significantly greater than placebo.

Anderson *et al*[9] compared the effectiveness of mefenamic acid and flufenamic acid, a related drug unavailable in this country, with a propoxyphene-acetaminophen combination (Darvocet N®, Wygesic®) in 30 patients. Twelve of the 30 patients preferred mefanamic acid, 10 preferred flufenamic acid, three preferred the propoxyphene-acetaminophen combination, and five had no preference or joint preferences.

Serious side effects from the drug therapy were not found in any of the above studies.

In conclusion, the prostaglandin inhibitors and antagonists, e.g., indomethacin, naproxen, and mefenamic acid appear to be significantly better than placebo in the treatment of dysmenorrhea, and may be more effective than aspirin in some women.

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Control of Drug Substitution Is the Real Issue

The complex anatomy of the drug substitution law in New Jersey shows the classic ailments of most regulatory measures in which government attempts to assume a role already —and best—filled by the expert practitioner.

Drug substitution, soon to be legally binding in New Jersey, is going to erode a time-proved system for providing drug therapy in favor of a new, allegedly "cost savings" scheme whose likely consequences will be a failure to save money and a lowering of health care standards now and in the future.

The editorial in the December 1978 issue of *The Journal* (75:895) excellently diagnosed this regulatory paradox. And the physician readers must have been struck by the irony of such a law.

Historically, by painstakingly removing the unknowns from the practice of medicine and related fields, health professionals and those who support their efforts have upgraded the quality and social benefit of health care in this country. Substitution, as proposed in New Jersey's law, on the other hand, introduces new risks into the prescribing and dispensing of medicine without any commensurate rewards for the consumer-patient or for society at large.

The legislative intent of the law, of course, is commendable: save consumers money without lowering the quality of drug therapy. But given the disappointing results in other states and the economic realities of the prescription marketplace, we cannot expect consumers to save appreciably on their drug bills. Numerous studies of substitution laws—in effect in other states since 1972—have failed consistently to document real savings to patients. And those price differences between brand-name drugs and their generic counterparts, heralded as being upward of 100 percent by substitution advocates, are starting to be recognized for what they are—inflated, politically contrived estimates.

Specifically, what are the problems inherent in substitution that make it a threat to patients and a problem for physicians? The editorial thoroughly covers most of them, but we would like to offer a few additional observations.

They deal with professional prerogatives, the knowledge which a physician has of his patient, variances in quality between and among drugs, and what we feel is one of the keys to better, more cost-effective health care in the future—the search for new drugs and medical devices. Substitution adversely affects all of these.

Professional Prerogative—In the past, traditional pharmacy laws and standards for drug product selection generally required some communication between the pharmacist and the physician before any change could be made in the original prescription. While the New Jersey law has the physician's "do not substitute" option, we question whether this will assure proper physician control over drug therapy.

In effect, the New Jersey law will change dispensing practices so that the physician really will not have an adequate opportunity to concur in the selection of a specific drug product. Since there is no direct provision for the physician to receive such notification, he or she will have no way of knowing precisely what product a patient receives from the pharmacist.

Effective, high-quality drug therapy obviously requires both knowledge of the drug and of the patient. Only the physician has both. Because a change in the source of the prescription can make a difference in treatment, and because the physician should have absolute control over all aspects of patient care, we believe it is irresponsible to alter the existing "checks and balances" of prescribing and dispensing.

By asking physicians to specify "do not substitute" on the prescription blank, legislators, and now health department officials, have increased the possibility that inadvertent and unreported substitutions will take place irrespective of the physician's intentions.

The real issue, then, is how to keep control in the hands of the physician. A legislative mandate that he or she justify the choice of medication is a subtle but an intentional attack on professional rights and a disruption of the doctor-patient relationship.

Drug Quality—Closely tied to this prerogative is the question of therapeutic equivalence among drug products. This question, of course, lies at the heart of product preference as a tangible, reliable means of assuring quality for the patient.

We believe physicians and pharmacists, despite formularies and federal assurances to the contrary, remain convinced, as they should, that there are differences in medical equivalence and quality among drug products. Source—the reputation and reliability of the manufacturer—remains the physician's assurance that he or she is prescribing only the highest quality medication. Cost cannot be the sole criterion for substitution. And we must all realize that limited geographical distribution of many non-branded generics and the suspect quality of other products—both brand and generic—critically limit choices for product selection.

The Food and Drug Administration's own recall and seizure records support the conclusions that some drug products on the market today do not produce the same therapeutic effect, nor are they all of the same high quality. For example, an analysis of FDA enforcement data conducted by Eli Lilly and Company in June 1978, shows that firms *not* spending great amounts on research (as compared to research-intensive firms) have seven times more FDA recalls and forty-three times more FDA-initiated court actions against them.

Consider, too, that the FDA applies different, less strin-

gent standards in approving "imitator" multisource drugs, has inadequate manpower to inspect regularly all drug manufacturing plants, and does not have the resources to test each and every formulation of a particular drug for therapeutic equivalence. For these reasons it is incorrect to assume that all manufacturers' formulations of the same drug will perform equally. Thus we believe that physicians will continue to prescribe and pharmacists to dispense drug products from sources they know and trust.

Substitution: A Disincentive to Continued Drug Research—Pharmaceutical research in this country is financed primarily from the sale of existing drugs. During the past five years American pharmaceutical firms have invested more than five billion dollars of their own funds in research and development (less than one percent comes from government). Those efforts have led to the introduction of several dozen innovations, each a significant contribution to improved health and reduced health care cost. Among the new drugs:

- A revolutionary treatment for duodenal ulcers
- A vaccine against pneumococcal pneumonia
- An agent to dissolve pulmonary blood clots
- Several improved anticancer compounds

For example, if every state should imitate New Jersey, and force the use of drugs made by firms which conduct no research, the research money will have to be found through increased prices for single-source products. That, in turn, will mean that rather than spreading the cost equitably among many hundreds of drug products, patients who need the latest drug will have to pay a disproportionate share of the cost of research.

There are other alternatives. Research could be reduced sharply, or even discontinued, or we could seek federal tax subsidies for it. We think the American people would oppose either of these dismal courses. The first would amount to scientific dereliction, and the second would bring more government into a field in which it has virtually no capabilities at a time when the consensus is that we already have too much government.

In sum, it is apparent that only minor savings are possible under the New Jersey approach. This and similar government schemes will tend to force increased prices for most single-source drugs, if essential company-financed pharmaceutical research is to continue.

Under the law physicians—if they make the effort—can maintain some control of drug product selection by writing on the prescription blank "do no substitute." They should at the very least do this much to maintain their prerogatives. This necessary veto power will allow the physician to work with the pharmacist in seeing that patients receive only the highest quality medication at the most reasonable price.

C. Joseph Stetler, President
Pharmaceutical Manufacturers Association

Catherine Pegues—MSNJ Auxiliary President



Catherine Pegues

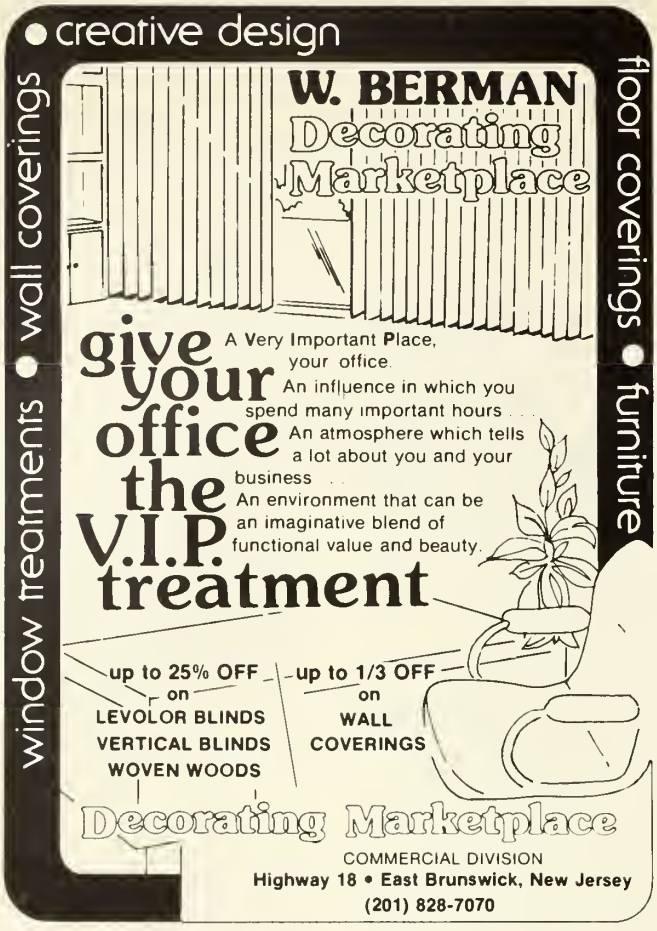
The 53rd President of the Medical Society of New Jersey Auxiliary is Catherine Pegues, wife of neurologist J. James Pegues.

Mrs. Pegues hails from Burlington County, where she served as president of that county society's auxiliary in 1971-1972. Born and raised in Terre Haute, Indiana, Cathy was graduated from the Northwestern University School of Speech in 1952, the same day her husband, Jim, was graduated from Northwestern Medical School.

Her interest in medical education and medical affairs was broadened by working as secretary and subsequently as administrative assistant to Dr. John P. Hubbard at the National Board of Medical Examiners in Philadelphia. She later was employed in the office of the Chief of Professional Services at the USAF Headquarters Hospital, Wiesbaden, Germany, for which she was given the USAF Distinguished Service Award.

Along with her involvement with the MSNJ Auxiliary, Cathy has a special interest in the Easter Seal Society, having served on the Burlington-Camden-Gloucester Board for over fifteen years. She also has served with the New Jersey Women's Committee for the Philadelphia Orchestra, the American Field Service, the Curtis Auxiliary to the Burlington County Memorial Hospital, and the Moorestown Home and School Association. For five years, Mrs. Pegues has been a volunteer director of plays in the Moorestown Middle School, using her talents in music as well as drama.

Cathy and Jim Pegues have two teenage sons and a married daughter. A.K.



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DOCTOR'S NOTEBOOK

Trustees' Minutes April 18, 1979

A regular meeting of the Board of Trustees was held on Wednesday, April 18, 1979, at the Executive Offices, Lawrenceville, New Jersey. Detailed minutes are on file with the secretary of your county society. A summary of significant actions follows:

HEW Region II Medicaid Meeting . . . Authorized reimbursement to Harvey J. Shwed, M.D., for expenses incurred while attending a two-day Region II Medicaid Meeting on Fraud and Abuse in New York (April 10-11).

Ruling on Amphetamines . . . Voted not to participate in a suit being brought by the Obesity and Nutrition Council to restrain the State Board of Medical Examiners from imposing upon physicians a restriction against prescribing and dispensing amphetamine and sympathomimetic amine drugs.

Note: MSNJ had assumed a position that amphetamines should be regulated and the State Board ruling is similar to Resolution #3 adopted by the 1978 House of Delegates.

National Health Insurance . . . Received the following information on the AMA's position on national health insurance adopted by that Board of Trustees on March 30, 1979:

1. That the AMA work toward amendment of S-760 and S-748 which will, as closely as possible, bring these pieces of legislation in conformity with Resolution 62 (I-78) and the recommendations of the Council on Legislation and the Council on Medical Service to the end that the AMA position on these consolidated amended bills would be one of endorsement or support of the legislation;

2. That lacking substantive amendments to these bills that would be agreeable to the Association, the AMA would again be in a position of opposition to the legislation;

3. That this action be communicated to the House of Delegates by the Board, with a statement of its reasons for the action and the amendments to these bills that the AMA will seek to achieve;

4. That the Board seek the assistance of the state, county, and medical specialty societies to effect this position;

5. That the Board indicate that it does not intend to call a special session of the House of Delegates at this time but calls attention to the mechanism whereby the House may call a special session if it chooses;

6. That a steering committee be appointed, composed of the Executive Committee of the Board and the Chairman and Vice-Chairman of the Council on Legislation and the Council on Medical Service to prepare the amendments and the communication to the House of Delegates and determine its timely distribution to the House.

House of Delegates Apportionment/1979 Dues Collection . . . Voted to follow past practice relative to 1979 dues collection and the determination of seating delegates from the various county societies at the May 1979 House of Delegates' meeting.

Note: According to the Bylaws, Chapter II, Section 1 (a), apportionment of delegates is determined by the Secretary of MSNJ in the interest of maintaining a total House membership not to exceed 400. Membership for 1979 had been projected at 8200, however, analysis revealed that as of April 2, 1979 there were approximately 6700 dues-paying members. Either there is a tremendous loss of members or the processing of dues is being delayed by the counties and/or physicians.

According to the Bylaws, Chapter II, Section 1 (c)—Delinquency—assessments are levied against the county societies, and if a county society has not paid for the registered number of physicians, the entire delegation of the county is declared delinquent and cannot be seated at the annual meeting. Traditionally, the basis for determining apportionment of delegates has been the number of members as of December 31 of the previous year. However, this does not employ the application of the Bylaws, which, if followed this year, would mean a loss of 55 delegates overall, using the standard of 22.5 members per delegate (projected 8200 members—delegates not to exceed 400).

The Board agreed that the Bylaws have not been adhered to in the past but expressed the opinion that the institution of a new policy without prior notice to the counties would not be advisable. The Speaker of the House indicated that the Bylaws are subject to more than one interpretation and believes there is no justification to change the established practice of the make-up of the House of Delegates for the May 1979 meeting. Mr. Maressa believes that the Bylaws are clear and not subject to several interpretations since the term "delinquent" can apply only to the current dues year.

Committee on Finance and Budget . . . Took the following actions on recommendations from the Committee on Finance and Budget which were suggested after consideration of the considerable drop in dues-paying members. There are a number of factors contributing to this:

1. There is a definite loss of membership.

2. There is a definite trend for more members to become dues-exempt or apply for and receive emeritus status (820 dues-exempt; 523 emeritus).

3. Delinquency is a serious problem (1380). These physicians have not resigned, died, or transferred, but they are not paying their dues or are having their dues withheld from MSNJ by the component society. (In prior years the delinquency rate ran between 500 and 600, most of whom would be reinstated by September of the dues year.) The situation must be corrected, and reduction of the delegates in MSNJ's House in proportion to the delinquency ratio of a given county is in order.

Recommendations—

(1) That the Committee on Finance and Budget and the Committee on Constitution and Bylaws be charged with the responsibility

of studying and developing Bylaw amendments that will provide the Medical Society of New Jersey with a sure and stable financial base and a collection system that will produce total collection of dues within the first quarter of a given dues year. **APPROVED.**

(2) That a special session of the House of Delegates be scheduled in the Fall of 1979 to act upon the Bylaw changes that are developed by this study and approved by the Board of Trustees. **APPROVED.**

. . . Approved a suggestion that the President of MSNJ and the Chairman of the Board of Trustees receive an annual stipend of $20,000. These amounts have been projected in the budget for 1979-80.

Note: This matter previously had been referred to the Committee by the Board of Trustees for consideration and recommendation.

1979-1980 Budget and Assessment . . . Approved the budget proposed by the Committee on Finance and Budget for submission to the House of Delegates.

. . . Approved the following recommendations:

1. That the 1980 dues be set at $200 per dues-paying member.

2. That a special assessment of $20 be levied for the New Jersey Foundation for Health Care Evaluations.

(Three members of the Board abstained from voting on Recommendation #2.)

Note: Assessments are projected on the basis of 7500 dues-paying members. In the event membership increases by September 1979, it is recommended that the House authorize the Board to reduce dues in conformity to the adopted budget and so notify the component societies.

MSNJ Student Association . . . Approved the following recommendations concerning the MSNJ Student Association and noted that the current membership stands at 187:

1. That the Board approve financial assistance for two students of MSNJSA to attend the AMA 75th Congress of Medical Education in Washington, D.C. on May 9-12, 1979.

2. That the outgoing and incoming officers of MSNJSA (8 students) be authorized to attend the inaugural reception and dinner during the MSNJ Annual Meeting.

Joint Practice Committee . . . Voted to accept the recommendation of the Executive Committee that the Board of Trustees approve the formation of a Joint Practice Committee to be composed of four members from MSNJ and four members from the New Jersey State Nurses Association.

. . . Approved the following goals and objectives of the Joint Practice Committee:

1. To address health care delivery issues from the perception of medicine and nursing.

2. To explore new patterns of practice toward improving patient care, such as joint practice, primary nursing, and the problem-oriented record.

3. To serve as a study panel to provide data concerning the attitudes of nurses and physicians about their respective roles and their practice patterns.

4. To provide a structure between the two professions to examine the increasing diversification of related allied health care personnel.

5. To address patterns of education of the two professions.

6. To explore issues arising from the joint practice of nurses and physicians.

Legislation . . . Approved the recommended positions on the following bills on which action had been deferred:

A-1321—Requires school boards to offer CPR as a mandatory course for all high school students. **CONDITIONAL APPROVAL**, provided the bill is amended for the course in CPR to be taught on a voluntary instead of mandatory basis.
A-1105—Requires physicians to secure specific written consent to elective surgical procedures which shall indicate the operating surgeon and identify any other participating physicians. The primary surgeon must be in continuous attendance unless an emergency occurs in which event his absence shall be noted in the operative record. The operative record which must be kept shall indicate the name, position and duties of each person in attendance at such operation. This record must be available for at least one year at both the hospital and the physician's office and available for the patient's inspection and reproduction. **ACTIVE OPPOSITION**, because this bill is not consistent with good medical care, fails to recognize sound concepts in teaching programs, ignores the existence of the surgical team, and does not demonstrate an awareness of the presence of the anesthesiologist and numerous assisting physicians.
S-515—Creates a Mental Health Study Commission consisting of 34 members to recommend to the Governor a Comprehensive plan for delivering mental health services in New Jersey. **ACTIVE OPPOSITION**, because there have been a series of commissions to study and evaluate the State's institutions, agencies, and services for the mentally ill and to develop a State plan to expand and improve the delivery of mental health services. The most recent findings having been published in 1976. Based on that experience, it appears that an appointed commission, inadequately financed, as proposed in this bill, cannot successfully undertake a highly skillful and impartial study and evaluation. We believe that an impartial study by trained persons outside of the direct State system of political and health care services is indicated and may be useful at this time. However, the format proposed in this bill, we believe, would be unproductive, a waste of taxpayers' funds and, therefore, not beneficial to the public.
S-517—To amend the Practicing Psychology Licensing Act in several respects concerning the membership on the Board, license fees and continuing education requirements. **ACTION DEFERRED**, pending further information from the New Jersey Psychological Association.
S-653—To provide for a procedure for the involuntary commitment of the mentally ill. **DISAPPROVED**, in favor of *A-475*.
A-708—Provides that patients must be discharged in an improved condition or in a condition of remission before being eligible for expungement proceedings. **APPROVED.**

. . . Deferred action on Assembly Bills 1110, 1111, 1112 which require Blue Shield and other health insurance contracts to require second opinions in all elective surgery as a precondition to payment, pending discussion of this item with the New Jersey Chapter of the American College of Surgeons at their meeting on April 27.

. . . Approved the recommended positions indicated on the following bills:

S-1297—Orechio—Electrologists Licensing Act

Authorizes the State Board of Medical Examiners to license electrologists. An electrologist is "a person who professionally removes hair from apparently normal skin of the human body by electrical, electronic, or other technical, scientific methods approved by the Board." The advisory board is to consist of three electrologists and three medical doctors, preferrably dermatologists. **APPROVED**

S-1511—Skevin—Controlled Dangerous Substances Therapeutic Research And Treatment

Creates a research program within the State Department of Health. Patients not responding to conventional therapy will be allowed into the program. Drugs currently on the Federal Drug Administration experimental list, i.e., marihuana, etc. will be utilized under Federal guidelines. The Medical Society of New Jersey will be given an active role, i.e., making recommendations to the Commissioner of Health for appointment to the Therapeutic Research and Treatment Qualification Review Board. **NO ACTION**

S-1517—Friedland—Payments of Attorneys' Fees in Civil Suits

Allows the Courts to award defense costs and attorneys' fees to successful defendants in situations where the court finds a professional liability claim or counterclaim to have been frivolous or repetitious or barred by the

Statute of Limitations. **ACTIVE SUPPORT**

S-3007—Feldman—Cardiopulmonary Resuscitation

Requires recreational and athletic facilities to have at least one CPR-trained employee on premises at all regular operating hours. **NO ACTION**

S-3017—Seardino—Medical Seviee Corporations

Lowers the statutory level of "groups" from 100 to 50 as was done previously with Blue Cross. In this fashion, parameters of the two plans are similar. It will not permit, however, a group rating formula which produces a rate higher than the community rate. **APPROVED**

S-3026—Merlino—Hearing Examination

Requires school systems to conduct an annual screening of hearing programs in accordance with Department of Education guidelines. (The Medical Society of New Jersey's Committee on Hearing and Speech supports this bill.) **ACTIVE SUPPORT**

S-3065—Maressa, Cafiero, et al—Medical Edueation

Appropriates 8 million dollars for acquisition, renovation, construction, in relation to the osteopathic facility in South Jersey. **ACTIVE OPPOSITION** because the Medical Society of New Jersey is convinced that the construction of a basic science facility in South Jersey is an unnecessary expenditure and misdirection of State funds designated for medical education.

S-3070—Hirkala—Certificate of Need

Would exempt free-standing bio-analytical laboratories, which deal primarily in interstate commerce from the certificate of need law. **NO ACTION**

S-3110—Scardino—Health and Safety of the Elderly

Provides a protection system centered upon reporting by professionals of instances of persons responsible for the custodial care of the elderly who have abused or exploited those left in their care.

Reports are to be made to the Office of the Ombudsman for the Institutionalized Elderly upon reasonable cause. The reports are to be confidential and the persons making such a report are granted civil and criminal immunity provided they have not acted in bad faith or with malicious purpose. **CONDITIONAL APPROVAL**, on the basis that the law should apply to everyone and not be limited to physicians. Further, the word "exploitation" should be more clearly defined.

A-1618—Muhler—Eyeglasses-Contact Lenses

Requires physicians and optometrists to supply patients with written lense prescriptions so they may purchase from the dispenser of their choice. **ACTIVE OPPOSITION**, to that portion of the bill referring to contact lenses, because MSNJ does not feel it would be beneficial to the health of the consumer. Contact lenses improperly used or fitted may cause irreparable damage to the cornea. The fitting of contact lenses should be done by the prescribing ophthalmologist or optometrist.

A-1682—Shapiro—Medical Edueation

Appropriates 2.15 million dollars to construct a 33-bed psychiatric hospital at CMDNJ-Newark. **NO ACTION** Law c. 138 ('78)

A-1686—Herman—Emergency Medieal Services

Extends the pilot program in mobile coronary intensive care units to June, 1979. **NO ACTION** Law c. 132 ('78)

A-1689—Burstein—Pharmaceutical Assistance to the Aged

Grants the Commissioner of Human Services regulatory control over the Pharmaceutical Assistance to the Aged Program in order to "control costs." **NO ACTION**

A-1763—Bassano—Controlled Dangerous Suhstances Research

Authorizes the Department of Health to conduct research into the efficacy of some drugs currently on the banned Schedule I list. **NO ACTION**

A-1779—Herman—Medicaid

Extends Medicaid eligibility to those currently not eligible if they encounter a catastrophic illness which means "any illness or injury requiring inpatient care in a hospital or skilled nursing facility, the costs of which exceed 25% of the person's annual net income in excess of the applicable categorical assistance eligibility level, whichever is less." **APPROVED**

A-1780—Lesniak—State Board of Medical Examiners

Provides the State Board of Medical Examiners may discipline physicians who charge excessive or unreasonable fees. **NO ACTION**

A-1851—Kavanaugh—Controlled Dangerous Suhstances Research

Same as S-1511 but restricts the products which may be used to marihuana and tetrahydrocannabinols and its derivatives. **NO ACTION**

A-1863—Maguire, Gallo—Medicaid Appropriations

Transfers six-million dollars from the general Medicaid budget and four-million dollars from the AFDC Program to the Pharmaceutical Assistance For the Aged Project. **NO ACTION**

A-1893—Bassano—Controlled Dangerous Suhstances Research

Creates a research program within the State Department of Health. Patients not responding to conventional therapy will be allowed into the program. Drugs currently on the Federal Drug Administration experimental list, i.e., marihuana, etc., will be utilized under Federal guidelines. The Medical Society of New Jersey will be given an active role, i.e., making recommendations to the Commissioner of Health for appointment to the Therapeutic Research and Treatment Qualification Review Board. **NO ACTION**

A-3003—Fortunato—Speeial Hospitals Emergency Services

All special hospitals would be required to provide full-time emergency services coverage. **ACTIVE OPPOSITION**, because the context of the bill is not clearly defined, i.e., (What is meant by resident physician?, etc.), plus the cost implication would defeat the purpose of the bill. There are twenty-two hospitals licensed as "special hospitals" within the State that have arrangements for a physician to be on call at all times, but they are not physically present.

A-3011—Sehwartz—CAT Scanners—Certifieate of Need

Requires that a certificate of need be secured for the operation of all CAT units. **ACTIVE OPPOSITION**, because the physicians in their private practices are currently exempt from the certificate of need and should continue to be so. Physicians do not enter into public fund raising, are not exempt from taxes, and are placing their own resources at risk. Further, they would be at a distinct disadvantage in trying to compete with hospitals for a certificate of need.

A-3014—Bassano—Marriage Lieenses

Requires all female applicants under 45 years of age for a marriage license to show laboratory evidence of a rubella response test as a condition precedent to the issuance of a marriage license. The physician is to inform the applicant of the medical significance of the results of the serological test. **NO ACTION**

A-3043—Bassano, Maguire—New Jersey Controlled Dangerous Suhstance Act

This bill permits duly authorized agents, officers or employees of an incorporated humane society to buy, possess, and use sodium pentobarbital to euthanize injured, sick, homeless, and unwanted dangerous pets or domestic or wild animals. **NO ACTION**

A-3103—Costello—Education

Grants parents the right to have their children excused from health, family, or sex education classes and also drug education courses on the basis of religious or moral beliefs. **NO ACTION**

Hospices . . . Approved the following recommendation from the Council on Public Relations concerning hospices:

That the Board of Trustees authorize representatives of the Council on Public Relations, to meet with representatives of the American Cancer Society and the New Jersey Hospital Association to discuss ways of educating physicians about the hospice concept.

Golden Merit Award Ceremony . . . Approved the following recommendation from the Council on Public Relations:

That the Board of Trustees attend the Golden Merit Award Ceremony and encourage the attendance of the presidents of specialty societies and the component societies since this is an important function recognizing physicians in the State who have served 50 years or more.

Note: Invitations also will be extended to the presidents of the county medical societies and the specialty societies and to the families of the recipients.

Epilepsy . . . Approved the following recommendation of the Council on Mental Health:

That the Board of Trustees acknowledge and support the national survey of epilepsy to be conducted by the RTI under the auspices of the National Institute of Neurologic and Communicative Disorders and Stroke of the National Institute of Health. The Council recommends, however, that the names of patients not be included on the information sheets to ensure confidentiality and protection of the patients.

Ad Hoc Committee on Emergency Medical Services Regionalization Proposal . . . Filed a report from the Ad Hoc Committee which contained a rec-

ommendation which, after approval by the Executive Committee, was forwarded to the Commissioner of Health and the chairman of the Health Care Administration Board:

That the Medical Society of New Jersey support the revised Emergency Medical Services Regionalization Regulations if amended to provide that the recognized segments of the medical profession existing on a statewide basis (MSNJ, the New Jersey Association of Osteopathic Physicians and Surgeons, and the Specialty Societies) be granted input into the development, the approval, and the implementation of the detailed plan for the various systems to be developed under the Emergency Medical Services Regionalization Project.

Note: On April 5 the Health Care Administration Board voted not to adopt the Emergency Medical Services regulations.

Nominating Committee . . . Received a report of the second meeting of the Nominating Committee (March 28, 1979), which revealed that the Committee had rescinded the actions taken at the February 14, 1979 meeting concerning the AMA Alternate Delegates and proceeded to vote on five candidates for the four vacancies. The first ballot recorded five candidates receiving a majority with a tie for the fourth and fifth positions, and on the second ballot one of the two received a majority. The proposed slate* is as follows: Howard D. Slobodien, M.D., Frank Y. Watson, M.D., Charles L. Cunniff, M.D., and James H. Spillane, M.D. Further action of the Committee was to place George L. Triebenbacher, M.D., in nomination for the Council of Mental Health from the Fourth District. The Nominating Committee then voted unanimously to request the Board of Trustees to appoint a parliamentarian to serve at each meeting of the Nominating Committee.

Emergency Medical Services . . . Voted to instruct the Committee on Emergency Medical Services to look into the matter of an emergency medical services plan for New Jersey.

Note: The Board observed that the Health Care Administration Board voted *not* to adopt the guidelines for the regionalization of emergency medical services; the Board of Trustees feels that a need still exists for some type of emergency medical plan for New Jersey.

*See *JMSNJ* 76:304 (April 1979) and 76:383 (May 1979)

†Awarded posthumously

Southern New Jersey Medical School . . . Approved a resolution from the Board of Trustees to the 1979 House of Delegates which resolved that a basic science facility in southern New Jersey should not be established at the present time. Subsequently the Board of Higher Education developed a position in conformity with MSNJ's position. The matter therefore has been withdrawn from the agenda of the House of Delegates.

Society of Anesthesiologists . . . Indicated to the chairman of the Council on Medical Services that the March 18 charge to that Council concerning the matter of economic inequity experienced by anesthesiologists intended that the Council commence looking for alternatives to the current handling of payment of physicians' services by third party payers so that once again the physician is in command. The basic issue is that non-government third party payers are vending physicians' services to the public at prices negotiated between the public and the insurance company without prior approval of the physician. The physician's current choice is either to accept the allowance or not deliver the services, unless a prior arrangement is made with the patient. Unless an alternative is found, physicians will find their services being sold at a price which bears no relation to the value of that service.

South Amboy Memorial Hospital Dispute . . . Agreed to support the medical staff of South Amboy Memorial Hospital to whatever extent is necessary in its dispute with the hospital brought about by a change in the bylaws, without the knowledge of the medical staff, whereby the officers of the medical staff shall be selected by the board of governors instead of the medical staff being permitted to choose its own representatives.

Summer Student Research Fellowship Program . . . Authorized an appropriation of $1,000 to support the Summer Student Research Fellowship of CMDNJ provided the student receives the total $1,000 fellowship.

Golden Merit Awards

The following members were recipients of MSNJ's Golden Merit Award (indicating fifty years of medical practice), presented at a special ceremony, followed by buffet luncheon, held in the new MSNJ headquarters building in Lawrenceville on May 24 and attended by family, friends, and colleagues of the awardees:

Atlantic County
Anthony Girard Merendino, M.D.
George Ruffin Stamps, M.D.

Bergen County
Paul Joseph Buckley, M.D.
Isador Gittelsohn, M.D.
Vernon Lee Hawes, M.D.
Frank S. Linane, M.D.
Romola Lyons, M.D.
Lester William Netz, M.D.
Charles Givens Prather, M.D.
Joseph Anthony Rubé, M.D.
Louis Minor Sosnow, M.D.
Gladys Claire Winter, M.D.

Burlington County
George Wallace Dickinson, M.D.

Camden County
Edwin Russell Ristine, M.D.
Edward A. Y. Schellenger, M.D.

Cumberland County
Charles Cunningham, M.D.

Essex County
Jack Richard Bevinetto, M.D.
Daniel M. Cerone, M.D.
George Mitchell Cohn, M.D.
Anthony F. DePalma, M.D.
Clement Harold Golden, M.D.
Morris Harris, M.D.
Theodore Stanley Heineken, M.D.
Meyer Jonas Kern, M.D.
Zelda Iris Marks, M.D.
Augustus John McKelvey, M.D.
Herman Paul Miller, M.D.
George Armin Paul, M.D.
John Vincent Reilly, M.D.
David Meyer Shor, M.D.
Charles Franklin Turner, M.D.
Harrison Rawlings Wesson, M.D.

Gloucester County
Leslie Thomas Sooy, M.D.

Hudson County
Edward N. Bookrajian, M.D.
Selig Leo Brauer, M.D.
Ralph Joseph Doran, M.D.
Henry Charles Fattel, M.D.
Charles Kemm Good, M.D.
Philip Greenberg, M.D.
Joseph Hillel, M.D.
Solomon Hirsch, M.D.
James Leonard Hollywood, M.D.
Charles Andrew Landshof, M.D.
Robert Bonser Lobban, M.D.
Irving Isidore Lynn, M.D.
Shafeek Nafash, M.D.
Albert Victor Saradarian, M.D.
Irving Schwarzwald, M.D.
Salomon Silvera, M.D.
†William Yudkoff, M.D.

Mercer County
Paul Louis Fabian, M.D.
Sydney Gene Fine, M.D.
Francis Augustin McGuigan, M.D.
Gerald Howard Miller, M.D.
Alfred Joseph Schwager, M.D.

Middlesex County
Charles Hiram Calvin, M.D.
Nathaniel Bertram Cole, M.D.
Howard E. Dieker, M.D.
Alexander Harry Fishkoff, M.D.
George Frigyes Frederick, M.D.
Estelle Elizabeth Kleiber, M.D.
Sollon M. Lazow, M.D.
Lawrence Howard Lief, M.D.
Leon Martin, M.D.
Morton R. Milsner, M.D.
Solomon Z. Nieman, M.D.

Monmouth County
Helen Dimitrow, M.D.
Charles C. Graves, M.D.
Marc Krohn, M.D.
James Percy Pregnall, M.D.
Abraham Rosenthal, M.D.

Morris County
Charles Hartranft Deichman, M.D.
Ruth Bridgman Ferriss, M.D.

Passaic County
Elias Joseph Hatem, M.D.
Herman Harold Holt, M.D.
Elias David Lawrence, M.D.
Michael Raab, M.D.
Abraham John Reinhorn, M.D.
William M. Sullivan, M.D.

Salem County
John Samuel Dunn, M.D.
Claude Bernard Mackes, M.D.

Sussex County
Dorsett Larew Spurgeon, M.D.

Union County
Hilde Baruch, M.D.
Joseph John Butenas, M.D.
Walter H. Cole, Jr., M.D.
Joseph Edward Franklin, M.D.
Merton Lyman Griswold, M.D.
Joseph Sadoff, M.D.
S. Lawrence Samuels, M.D.
Stanley Tolor, M.D.

CMDNJ Notes*

Stanley S. Bergen, Jr., M.D.
President

Throughout the state, there has been growing evidence of the College's interest and involvement in reaching out to New Jerseyans with programs that highlight preventive medicine and good health habits. As a prime example, this month marks the first anniversary of the appearance of the weekly "to health" column in a dozen daily newspapers and twice that many weekly newspapers circulating in practically all counties in the state. The column is appearing also in a few publications issued by community hospitals and industrial firms.

This material has been responsible for the dissemination of vital health information to many New Jerseyans who otherwise would not be getting it. It is written with the cooperation and over the byline of faculty members at all schools of CMDNJ, and it is distributed and promoted by the CMDNJ Public Affairs Department under a grant from the Hunterdon Health Fund. Some of the topics covered during its first year include hyperactivity in children, stress, the menopause, depression, jogging, the use of hypnotism in psychotherapy, and sleep disorders.

Readers' queries are invited, and since the column first appeared, hundreds of phone calls and letters have been received. All are answered, either in print or individually, by the office of Public Affairs after contacting the physician concerned.

A surprise benefit, from the public relations standpoint, has been the appearance of excerpts in several popular women's magazines and in nationally syndicated health columns, thus helping to give CMDNJ recognition beyond the state's boundaries.

Another successful program funded by the Hunterdon Health Fund has been "Health Education Day," observed each May 10 since 1976 to commemorate the dedication of the CMDNJ Newark campus. This year's observance had a double benefit, for it was also the date of the dedication of the CMDNJ College Hospital in Newark.

Schools, public libraries, pharmacies, hospitals, health institutions, and religious and community groups received packets of resource materials and information on conducting programs, arranging displays, and exhibiting posters on health-related topics as part of their participation in "Health Education Day." In addition, CMDNJ faculty members, students, and employees conducted "Health Education Day" activities on two campuses—the CMDNJ-Rutgers Medical School in Piscataway and the Newark campus. Hundreds of high school students with an expressed interest in health sciences were taken on tours of the campuses and shown exhibits and displays prepared by faculty and students of the various CMDNJ schools and units.

CMDNJ's Office of Consumer Health Education and Public Affairs Department has carried out a statewide informational and educational program to alert New Jerseyans that "Good Health Is Up to Me." This theme emphasized to all residents of New Jersey to take greater responsibility for their own health care by establishing sound health habits.

Another example of CMDNJ's involvement in the community's health, particularly in South Jersey, has been the Camden Area Health Education Center (AHEC) program, which is now halfway through its first-year planning stage. As reported in the January issue of *The Journal* (p. 61), this unique project in health care and education for southern New Jersey is now focusing on the development of details such as formal agreements between CMDNJ and the participating community hospitals, the appointment of a Program Advisory Committee, and the establishment of the program's organizational and management structure.

Report from the Foundation

Daniel J. O'Regan, M.D.
Medical Director

In response to several requests, a brief glossary of some of the terms and acronyms which appear in this space is hereby provided:

1. **Professional Standards Review Organization (PSRO)**—Eight in New Jersey. Federally funded; physician-operated. To evaluate the quality, necessity, and appropriateness of medical care to federal patients (Medicare, Medicaid; Maternal and Child Health).

2. **Professional Standards Review Council (PSRC)**—Federally funded. Physician members represent all PSROs. Also has public members. Coordinates PSRO activities. Reviews appeals from PSRO decisions, plus other duties.

3. **Health Care Financing Administration (HCFA)**—Part of DHEW. Incorporates Medicare, Medicaid, PSRO, and other functions.

4. **Health Standards and Quality Bureau (HSQB)**—Part of HCFA. Oversees PSRO program.

5. **Office of PSROs (OPSRO)**—Under HSQB. Has direct responsibility for PSRO operations.

6. **National Professional Standards Review Council (NPSRC)**—Composed of 11 physicians. Advisory to the Secretary, DHEW on all matters concerning PSRO program.

7. **American Association of Professional Standards Review Organizations (AAPSRO)**—Private organization. Physician-controlled. Interested in proper implementation of PSRO as a quality assurance mechanism.

8. **Health Systems Agency (HSA)**—Federally funded. Five in New Jersey. Responsible for developing plan for health services in respective areas. Involved in Certificate of Need (CON) process.

9. **Health Maintenance Organization (HMO)**—Prepaid health organization. Privately operated, subject to state and federal regulations. Offers medical services to subscribers on prepaid basis. Promotes efficiency through ambulatory services with reduction in overuse of hospital beds.

10. **Individual Practice Association (IPA)**—A variety of HMO which uses physician members working in their own offices. Does not require construction of facilities, and permits freedom of choice of physician and patient.

11. **American Association of Foundations for Medical Care (AAFMC)**—Private organization, composed of physicians interested in all aspects of medical care delivery. IPA concept grew out of Foundation for Medical Care (FMC) experience.

12. **Diagnosis Related Groups (DRGs)**—Statistical grouping of diagnosis by "case-mix" and intensity of services. Study being undertaken by NJ Department of Health. Will probably be part of prospective rate-setting mechanism for New Jersey hospitals.

Physicians Seeking Location in New Jersey

The following physicians have written to the Executive Office of MSNJ seeking information on possible opportunities for practice in New Jersey. The information listed below has been supplied by the physician. If you are interested in any further information concerning these physicians, we suggest you make inquiries directly to them.

ALLERGY—Yalamanchi K. Rao, M.D. 354 Henry Street. Apt. #2, Brooklyn, NY 11201. Nagpur Medical College (India) 1968. Board eligible. Group, partnership, solo, institutional, research. Available July 1979.

Richard W. Huss, M.D. 555 Graham Road, Fort Sam Houston, TX 78234. CMDNJ 1971. Subspecialty, internal medicine. Board certified (IM). Group or solo. Available September 1979.

Andrew W. Green, M.D., 1 Raintree Island, Apt. 6, Tonawanda, NY 14150. St. Louis University 1974. Subspecialty, internal medicine. Board certified (IM). Solo or group. Available July 1979.

ANESTHESIOLOGY—Ajit V. Pai, M.D., 8136 Braeburn Terrace, Indianapolis, IN 46219. Topiwala National (India) 1973. Board eligible. Group or partnership. Available September 1979.

Mohammed A. Mohiuddin, M.D., 104 West Franklin Street, Apt. 908, Richmond, VA 23220. Osmania Medical College (India) 1971. Board eligible. Group or partnership. Available July 1979.

Kie C. Ohn, M.D., 520 Second Avenue, Apt. 11-F, New York, NY 10016 Seoul National (Korea) 1971. Subspecialty, family practice. Board eligible. Group, partnership, or solo. Available July 1979.

Vinod Malhotra, M.D., 40 Prospect Avenue, Apt. 63, Hackensack 07601. University of Delhi (India) 1972. Board eligible. Group, partnership, or solo. Available July 1979.

Hyun Hwang, M.D., 435 East 70th Street, Apt 7-F, New York, NY 10021. Yonsei University (Korea) 1964. Board eligible. Group, partnership, solo, or industrial. Available July 1979.

Subbarao Narepalem, M.D., 2947 Northview Drive, Youngstown, OH 44504. Andhra Medical College (India). Board eligible. Group, partnership, fee for service. Available July 1979.

Subba Rao Chennupati, M.D., 407 Lindsay Court #12, Louisville, KY 40206. Rangaraya (India) 1970. Board eligible. Group, partnership, solo. Available July 1979.

CARDIOVASCULAR DISEASES—Il Young Kim, M.D., 7044 Glen Meadow Lane, Cincinnati, OH 45237. Seoul National University (Korea) 1971. Subspecialty, internal medicine. Board certified (IM). Group, partnership, institutional. Available July 1979.

Stephen M. Barolsky, M.D., 6347 Caton Street, Pittsburgh, PA 15217. SUNY—Downstate 1973. Subspecialty, internal medicine. Board certified (IM). Group, partnership, institutional, research. Available July 1979.

Rajinder K. Shalla, M.D., 353 East 17th Street, Apt. 8-F, New York, NY 10003. All-India Institute (India) 1968. Subspecialty, internal medicine. Board eligible (IM). Group, institutional. Available July 1979.

Vrajesh M. Parikh, M.D., 2520 South King Drive, Chicago, IL 60616. B.J. Medical College (India) 1970. Subspecialty, internal medicine. Board eligible (IM). Group, partnership, research. Available July 1979.

Shashibalao P. Sehgal, M.D., Rockefeller University, 1230 York Avenue, New York, NY 10021. Seth G.S. Medical College (India) 1973. Subspecialty, internal medicine. Board certified (IM). Group, partnership, public health, industrial, institutional. Available July 1979.

DERMATOLOGY—Fred V. Kemp, M.D., 114 East Bailey Road, Naperville, IL 60540. St. Louis University 1973. Group, partnership, or solo. Available July 1980.

EMERGENCY MEDICINE—Nicanor C. Lopez, M.D., 1432 Arch Street, Apt. 8202, Norristown, PA 19401. Far Eastern University (Philippines) 1967. Subspecialty, general practice. Group, partnership, emergency room. Available July 1979.

ENDOCRINOLOGY—George Tun-I Yang, M.D., 1764 Bising Avenue, #2, Cincinnati, OH 45239. Taipei Medical (Taiwan) 1972. Subspecialty, internal medicine. Board certified (IM). Group, partnership, solo. Available July 1979.

FAMILY PRACTICE—Timothy B. Eckel, M.D., 1410 Bloom Road, Danville, PA 17821. Temple 1976. Board eligible. Group or partnership. Available July 1979.

GASTROENTEROLOGY—Alan M. Nelson, M.D., 41B Monroe Street, Milford, CT 06460. Georgetown 1974. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

Robert S. Goldblatt, M.D., 150 Division Street, Derby, CT 06418. George Washington University 1974. Subspecialty, internal medicine. Board eligible (IM). Group or partnership. Available July 1979.

Jon David Green, M.D., 509 Fairview Avenue, Apt. C-1, Orange 07050. CMDNJ 1974. Subspecialty, internal medicine. Board certified (IM). Group, partnership, solo. Available July 1979.

Hua-Lung Lin, M.D., 911 West Apollo Avenue, Tempe, AZ 85283. National Taiwan University 1972. Subspecialty, internal medicine. Board eligible (IM). Group, partnership, solo. Available July 1979.

Myung Woong Kim, M.D., 6582 Poplar Woods, Circle S., Germantown, TN 38138. Yonsei University (Korea) 1968. Subspecialty, gastroenterology. Board certified (IM). Group, partnership, solo. Available July 1979.

Vithal Kusuma, M.D., 1227 S. Harlem Street, Berwyn, IL 60402. Osmania (India) 1969. Subspecialty, internal medicine. Board certified (IM). Group or partnership. Available July 1979.

Ira M. Litzenblatt, M.D., 4B Staunton Court Road, Farmington, CT 06032. Michigan 1975. Subspecialty, internal medicine. Board certified (IM). Group, partnership, associate. Available July 1980.

GENERAL PRACTICE—Dharamsi D. Shah, M.D., 22-4B Passaic Avenue, Nutley 07110. Grant Medical College (India) 1974. Subspecialty, psychiatry. Board eligible (psych.). Group, partnership, solo. Available July 1979.

James W. Warren, M.D., Ainsworth Health Clinic, Fort Hamilton, Brooklyn, NY 11252. Meharry 1973. Group, partnership, rural sponsorship, emergency room. Available.

Sam W. Law, M.D., 60 Eldridge Street, Apt. 6, New York, NY 10002. National University (Taiwan) 1970. Group, partnership, solo. Available.

HEMATOLOGY—Ming-Swang Lok, M.D., 163 Oxford Avenue, Buffalo, NY 14209. National Defense Medical Center (Taiwan) 1970. Subspecialty, internal medicine. Board certified (IM). Research or institutional. Available July 1979.

Subhash C. Proothi, M.D., 86-D Village Green North, East Providence, RI 02915. Rohtak Medical College (India) 1970. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

INFECTIOUS DISEASES—James E. Milder, M.D., 870 Johnsdale Drive, Lexington, KY 40505. Temple 1974. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

INTERNAL MEDICINE—Franciska Katona, M.D., Village Lane Apts., Oak-2, Abington, PA 19001. Temple 1976. Board eligible. Industrial, occupational, research, clinic, preventive medicine. Available July 1979.

Tsue-Pin Lin, M.D., 1945 Corlies Avenue, Neptune 07753. Kaohsiung (Taiwan) 1971. Subspecialty, general practice. Board eligible. Group, partnership, or solo. Available July 1979.

B. Madhusudhan Reddy, M.D., 69 Gold Street, Apt. 12-C, New York, NY 10038. Osmania Medical College (India) 1974. Board eligible. Group, partnership, institutional. Availavle July 1979.

Indu B. Patel, M.D., 9668 Dundawan Road, Baltimore, MD 21236. B.J. Medical College (India) 1971. Board eligible. Group, partnership, institutional. Available July 1979.

Rangachari Srinivasan, M.D., 1325 Pennsylvania Avenue, Apt. 15-D, Brooklyn, NY 11239. Stanley Medical College (India) 1970. Subspecialty, neoplastic diseases. Board certified. Group, partnership, solo. Available July 1979.

Mohamed Ziauddin, M.D., VA Center, Martinsburg, WV 25401. Madurai Medical College (India) 1969. Board eligible. Group, partnership. Available July 1979.

Robert I. Chapnick, M.D., 98-01 67th Avenue, Apt. 12-U, Forest Hills, NY 11374. SUNY—Downstate 1976. Subspecialty, emergency medicine. Board eligible. Group or partnership. Available August 1979.

Bui Ngoc Diep, M.D., 417 NE 13th Street, Oklahoma City, OK 73104. Faculty of Medicine (Vietnam) 1971. Subspecialty, neoplastic diseases. Board eligible. Group, partnership, institutional. Available July 1979.

Imdad H. Butt, M.D., Apt. 5B-1 Franklyn Manor 114 Franklyn Street, Morristown 07960. King Edward (Pakistan) 1970. Subspecialty, general practice. Board eligible (pediatrics). Partnership or solo. Available July 1979.

John S. Smiarowski, M.D., U.S. Naval Hospital, 7500 East Carson Street, Long Beach, CA 90822. Yale 1971. Subspecialty, gastroenterology. Board certified. Group, partnership, solo. Available August 1979.

Fred H.Miller, M.D., 378 Valley Park South, Bethlehem, PA 18018. Jefferson 1975. Board eligible. Group, partnership, solo. Available October 1979.

Rachakonda D. Prabhu, M.D., 80-15 41st Avenue, Apt. 4C8, Elmhurst, NY 11373. R. G. Kar Medical College (India) 1971. Subspecialty, pulmonary diseases. Board certified. Group, partnership, solo, or institutional. Available July 1979.

Samala R. Swamy, M.D., 355 Bard Avenue, Apt. 32, Staten Island, NY 10310. Kakatiya Medical College (India) 1972. Subspecialty, cardiovascular diseases. Board certified. Group, solo, or institutional. Available July 1979.

Jay H. Alexander, M.D., 30 Waterside Plaza, Apt. 9-E, New York, NY 10010. Loyola 1976. Subspecialty, emergency medicine. Board eligible. Industrial, emergency room, school health, institutional. Available July 1979.

Joel S. Steinberg, M.D., 6641 Oakland Street, Philadelphia, PA 19149. Temple 1976. Subspecialty, family practice. Board eligible. Group or partnership. Available July 1979.

N. Satish Kamath, M.D., 401 Tobin Drive, #310, Inkster, MI 48141. Karnatak Medical College (India) 1972. Board eligible. Group, partnership, solo. Available August 1979.

Sambaiah Kankanala, M.D., 122 Ashland Place, Apt. 11-N, Brooklyn, New York 11201. Osmania Medical School (India) 1972. Subspecialty, pulmonary diseases. Board certified. Group or solo. Available July 1979.

Nadakavil M. Koshy, M.D., 8 Slate Creek Drive, Apt. 9, Cheektowaga, NY 14227. Kottayam Medical College (India) 1971. Subspecialty, cardiovascular diseases. Board certified. Group, partnership, solo, or institutional. Available July 1979.

Chakumkal Cyriac,M.D., 3001 South Hanover Street, Baltimore, MD 21230. T.D. Medical College (India) 1973. Subspecialty, general practice. Board eligible. Group, partnership, solo. Available August 1979.

George T. Yang, M.D., 1764 Bising Avenue, Apt. 2, Cincinnati, OH 45239. Taipei (Taiwan) 1972. Subspecialty, endocrinology. Board certified. Group, partnership, solo, or emergency room. Available July 1979.

Pervaiz Rahman, M.D., 1650 Grand Concourse, Bronx, NY 10457. Dow Medical College (Pakistan) 1972. Subspecialty, gastroenterology. Board eligible. Group, partnership, institutional. Available July 1979.

Jambur E. Chandrashekar, M.D., 155 Forest Road, #39, St. John's A1A 1E7, Newfoundland, Canada. Mysore Medical College (India) 1970. Board eligible. Group, partnership, solo, or institutional. Available July 1979.

Ashok K. Shetty, M.D., 552 Amberson Plaza, Six Bayard Road, Pittsburgh, PA 15213. Government Medical (India) 1970. Board eligible. Partnership, solo. Available October 1979.

Soma N. Pulipati, M.D., 725 East Main Street, Kings Park, NY 11754. Osmania (India) 1971. Subspecialty, cardiology. Board certified. Solo, group, partnership (in that order). Available July 1979.

Richard E. Handelsman, D.O., 40 Prospect Avenue, Apt. 3-M, Bldg. 1, Norwalk, CT 06850. College of Osteopathic Medicine & Surgery 1976. Group or partnership. Available August 1980.

Charles F. McNally, M.D., Box 1774, 61 Moulton Road, Duxbury, Massachusetts 02332. New York Medical College 1968. Board certified. Group or partnership. Available July 1979.

Carol D. Silver, M.D., 1619 Third Avenue, Apt. 4-G, New York, NY 10028. Duke 1970. Subspecialty, medical oncology. Board certified. Solo, group, or partnership. Available August 1979.

Muhammad Tayyab, M.D., 2112 Starling Avenue, Apt. 4-L, Bronx, NY 10462. King Edward (Pakistan) 1972. Board eligible. Salaried, solo, group. Available November 1979.

NEOPLASTIC DISEASES—Victor D. Ribeiro, M.D., 82 Beacon Hill, Apt. 1B3, Dobbs Ferry, NY 10522. Goa Medical College (India) 1970. Subspecialty, internal medicine. Board certified (IM). Group or institutional. Available July 1979.

Gregory P. Burke, M.D., 45 Viscount Road,Longmeadow, MA 01106. SUNY—Downstate 1972. Subspecialty, internal medicine. Board Certified (IM). Group or academic. Available.

NEUROLOGY—Hungi Papanna Eswara, M.D., 843 School Lane House, 5450 Wissahickon Avenue, Philadelphia, PA 19155. Government Med. College (S. India) 1971. Board eligible. Solo, group, partnership. Available July 1979.

OBSTETRICS/GYNECOLOGY—Devendra M. Bhardwaj, M.D., 242 Gurdon Street, Bridgeport, CT 06606. S.M.S. Medical College (India). Board eligible. Group, partnership, solo, HMO. Available July 1979.

Kenneth Garrett, M.D., 31-A1 Lakeshore Drive, Farmington, CT 06032. NYU 1975. Board eligible. Group, partnership. Available August 1979.

Charles Ackerman, M.D., 353 East 17th Street, New York, NY 10003. Tel Aviv (Israel) 1975. Board eligible. Group or partnership. Available September 1979.

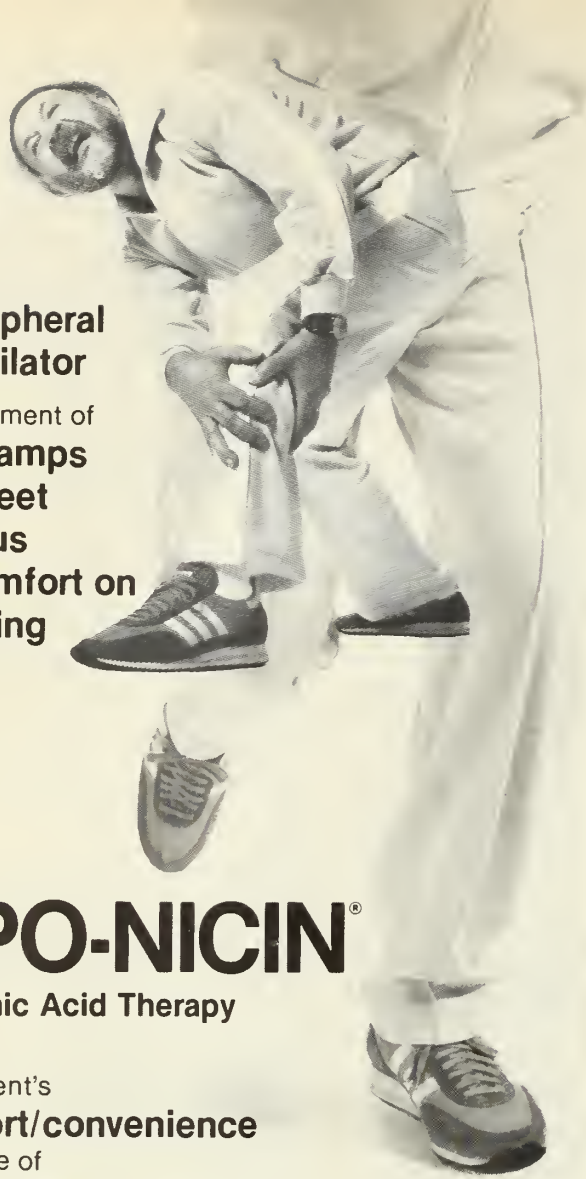
Kok C. Chang, M.D., 411 West 114th Street, Apt. 4-B, New York, NY 10025. National Taiwan University 1973. Board eligible. Group, partnership, solo. Available August 1979.

Sang Bak Yoo, M.D., 10856 Nottingham, Detroit, MI 48224. Kyung-Pook University (Korea) 1969. Board eligible. Group, partnership, or solo. Available July 1979.

Steven B Wirts, M.D., 340 Nina Street, New Windsor, NY 12550. Jefferson 1974. Board eligible. Group or partnership. Available July 1979.

Heinz O. Osterholzer, M.D., PSC 451, K.I. Sawyer AFB, Michigan 49843. Hahnemann 1968. Board eligible. Group or partnership. Available June 1980.

OCCUPATIONAL MEDICINE—Arthur E. Mittnacht, M.D., 299 Edgerstoune Road, Princeton 08540. Columbia 1945. Subspecialty, orthopedic surgery. Board certified (orthopedic surg.) Industrial,



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Each yellow tablet contains:
Nicotinic Acid 250 mg.
Niacinamide 75 mg.
Ascorbic Acid 150 mg.
Thiamine HCL (B-1) 25 mg.
Riboflavin (B-2) 2 mg.
Pyridoxine HCL (B-6) 10 mg.
DOSE: 1 to 3 tablets daily.
AVAILABLE: Bottles of 100, 500.

LIPO-NICIN®/100 mg.

Each blue tablet contains:
Nicotinic Acid 100 mg.
Niacinamide 75 mg.
Ascorbic Acid 150 mg.
Thiamine HCL (B-1) 25 mg.
Riboflavin (B-2) 2 mg.
Pyridoxine HCL (B-6) 10 mg.
DOSE: 1 to 5 tablets daily.
AVAILABLE: Bottles of 100, 500.

Indications: For use as a vasodilator in the symptoms of cold feet, leg cramps, dizziness, memory loss or tinnitus when associated with impaired peripheral circulation. Also provides concomitant administration of the listed vitamins. The warm tingling flush which may follow each dose of LIPO-NICIN® 100 mg. or 250 mg. is one of the therapeutic effects that often produce psychological benefits to the patient.

Side Effects: Transient flushing and feeling of warmth seldom require discontinuation of the drug. Transient headache, itching and tingling, skin rash, allergies and gastric disturbance may occur.

Contraindications: Patients with known idiosyncrasy to nicotinic acid or other components of the drug. Use with caution in pregnant patients and patients with glaucoma, severe diabetes, impaired liver function, peptic ulcers, and arterial bleeding.

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REFER TO PDR

school health, emergency room. Available.

OPHTHALMOLOGY—Jonathan F. Wise, M.D., 1175 York Avenue, Apt. 4-H, New York, NY 10021. SUNY—Buffalo 1973. Subspecialty, pediatrics. Board eligible. Group or partnership. Available.

Joshua Frankel, M.D., 161 North Sycamore Avenue, Los Angeles, CA 90036. Maryland 1971. Board certified. Partnership, solo, institutional. Available.

Alan I. Mandelberg, M.D., 284 Wintergreen Way, Rochester, NY 14618. SUNY—Buffalo 1972. Board certified. Group, partnership, or solo. Available July 1979.

Raphael J. Sapeika, M.D., 7 Windsor Road, Great Neck, NY 11021. Cape Town (South Africa) 1970. Board eligible. Group or partnership. Available July 1979.

Stephen L. Bosniak, M.D., 782 Westend Avenue, Apt. 63, New York, NY 10025. Georgetown 1973. Board elilible. Group, partnership, solo, institutional, or industrial. Available.

ORTHOPEDICS—Upendra Kumar Sinha, M.D., 86-16 60th Avenue, Apt. 2-E, Rego Park, NY 11373. Prince of Wales (India) 1968. Board eligible. Group, partnership, solo. Available July 1979.

OTOLARYNGOLOGY—Vijay Chandra Singh Bais, M.D., 2174 Green Spring Drive, Kettering, OH 45440. Mahatma Gandhi (India) 1961. Board certified. Solo, partnership, group. Available.

OTORHINOLARYNGOLOGY—Jang-Huei Jang, M.D., 7563 Drumheller Drive, Richmond, Virginia 23225. Chung-Shan (Taiwan) 1967. Board eligible. Group or solo. Available July 1979.

Gregory J. Milmoe, M.D., 259 Canner Street, New Haven, CT 06511. Chicago 1973. Board eligible. Group, research, partnership. Available August 1979.

PATHOLOGY—Dhanasaran Mongkolsmai, M.D., 900 West Spencer Street, Apt. 302, Philadelphia, PA 19141. Ramathibodi (Thailand) 1971. Special interest, clinical pathology. Board certified. Group, partnership, institutional. Available September 1979.

Ahm Abdul Mannan, M.D., 215 East 77th Street, Apt. 3-C, New York, NY 10021 Dacca (Pakistan) 1961. Special interest, clinical pathology. Board eligible. Group, research, institutional. Available July 1979.

Luis R. Molinas, M.D., 3041 West Chester Pike, Apt. G-7, Broomall, PA 19008. National University (Paraguay) 1972. Board eligible. Partnership, group, institutional. Available July 1979.

Ruly Yoediono, M.D., 10304 East Malcolm Circle, Cockeysville, MD 21030. Padjadjaran (Bandung) 1971. Special interest, clinical pathology. Board eligible. Group, partnership. Available September 1979.

Mehboob M. Fatteh, M.D., 3405 Crane Ferry Road, Augusta, GA 30907. H.H.L. Medical College (India) 1968. Board eligible. Group, partnership, solo, institutional. Available March 1979.

Frank R. Rudy, M.D., 7614 Woodcrest Avenue, Philadelphia, PA 19151. Pit-

tsburgh 1974. Board eligible. Institutional, partnership, research. Available July 1979.

Punita P. Kothari, M.D., 5990 Henry Avenue, Apt. 4, Philadelphia, PA 19128. M. P. Shah (India) 1969. Board eligible. Associate, group, partnership, institutional. Available July 1979.

V. A. Kagali, M.D., 54 Denby Acres, 7426 Henry Clay Boulevard, Liverpool, NY 13088. Bangalore (India) 1971. Board certified. Group, partnership. Available.

PEDIATRICS—Georgino R. Reyes, M.D., 18 Palmetto Circle, Brownsville, TX 78521. Santo Domingo (Dominician Republic) 1960. Board certified. Group, partnership, research. Available July 1979.

Jesse M. Mintz, M.D., 265-24 74th Avenue, Apt. G-1, Glen Oaks, NY 11004. Columbia 1972. Board certified. Institutional, group. Available July 1979.

Ganesh N. Deshpande, M.D., 49 Stoneledge Court, Williamsville, NY 14221. Grant Medical College (India) 1970. Subspecialty hematology. Board eligible. Institutional, research, group. Available July 1979.

Chalermlarp Mongkolsmai, M.D., 900 West Spencer Street, Apt. 302, Philadelphia, PA 19141. Ramathibodi Medical School (Thailand) 1971. Subspecialty, pediatric cardiology. Board certified. Institutional, research, group. Available September 1979.

Nam Soo Lee, M.D., Meadowbrook Apt. 44, Blessing Road, Slingerlands, NY 12159. Seoul National University (Korea) 1971. Subspecialty, pediatric cardiology. Board eligible. Solo, group, research. Available July 1979.

Elias G. Gouel, M.D., 6036 East Pratt, Baltimore, MD 21224. Ain Shams University (Egypt) 1971. Board eligible. Group, partnership. Available August 1979.

Ira B. Toback, M.D., 4618 Avenue J. Brooklyn, NY 11234. Louvain (Belgium) 1976. Board eligible. Partnership or group. Available July 1979.

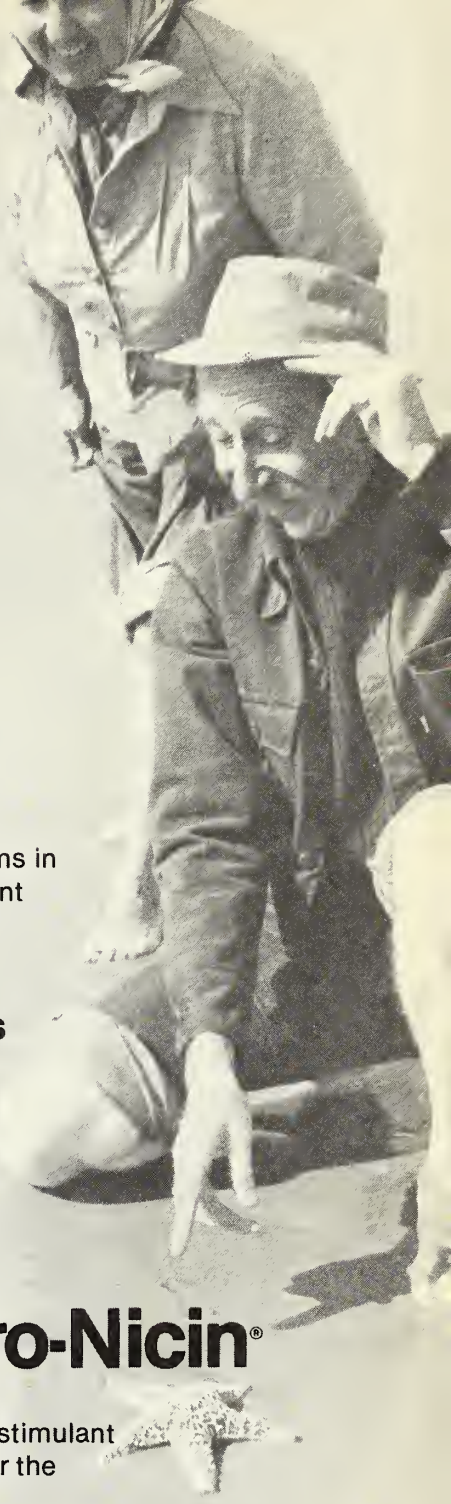
Boo Woong Lee, M.D., 1160 Midland Avenue, Apt. 8-M, Bronxville, NY 10708. Yonsei (Korea) 1973. Board eligible. Group, partnership, solo, institution. Available July 1979.

Roy Gomez, M.D., 300 Lookout Avenue, Apt. D-9, Hackensack 07601. Trivandrum (India) 1971. Board eligible. Group, partnership, institution, solo. Available July 1979.

L. N. Gajula, M.D., 3318 Borden Apts., Third Avenue, Long Branch 07740. Osmania (India) 1970. Board eligible. Group, partnership, solo. Available July 1979.

PHYSICAL MEDICINE/REHABILITATION—Diane C. Griffin, M.D., 29 East 29th Street, New York, NY 10016. University of Rome (Italy) 1974. Subspecialty, general practice Board eligible. Institutional, group, research. Available July 1979.

PSYCHIATRY—Sumanlal J. Kaneria, M.D., P.O. Box 9587, Cheyenne, WY 82001. M.P. Shah Medical School (India) 1971. Board eligible. Academic type practice. Available October 1979.



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Riboflavin	2 mg.
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AVAILABLE: Bottles 100, 500, 1000

SIDE EFFECTS: Most persons experience a flushing and tingling sensation after taking a higher potency nicotinic acid. As a secondary reaction some will complain of nausea, sweating and abdominal cramps. The reaction is usually transient.

INDICATIONS: As a cerebral stimulant and vasodilator.

RECOMMENDED GERIATRIC DOSAGE: One capsule three times daily adjusted to the individual patient.

WARNING: Overdosage may cause muscle tremor and convulsions.

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Balasubramaniam Gulasekaram, M.D., Apt. 1-D, Beechwood Hall, Westchester City Medical Center, Valhalla, NY 10595. Colombo (Sri Lanka) 1970. Board eligible. Research, institutional, group. Available September 1979.

PULMONARY DISEASES—Joseph I. Matthews, M.D., OSC Box 553, Walter Reed Hospital, Washington, DC 20012. Creighton 1969. Subspecialty, internal medicine. Board certified (IM). Group, institutional, partnership. Available August 1979.

Richard T. Bell, M.D., 1740 Aidenn-Lair Road, Dresher, PA 19025. Jefferson 1972. Subspecialty, internal medicine. Board certified (IM). Group, solo. Available July 1979.

Harjinder S. Gogia, M.D., 6201 North 10th Street, Apt. 435, Philadelphia, PA 19141. Punjabi University (India) 1973. Subspecialty, internal medicine. Board certified (IM). Group, institutional. Available July 1979.

RADIOLOGY—Seetharam A. Mattupalli, M.D., 7083 Middle Valley Walk, Apt. B, St. Louis, MO 63123. Guntur (India) 1971. Subspecialty, diagnostic radiology. Board eligible. Partnership, research, group, administrative, institutional. Available July 1979

Henry Mang, M.D., 8 Predmore Avenue, Colonia 07067. Sydney (Australia) 1965. Subspecialty, nuclear medicine. Board certified. Any type practice. Available.

Patrick F. Zazzaro, M.D., 6839 Shawnee Road, Richmond, VA 23225. CMDNJ-Rutgers 1975. Particular interest—diagnostic radiology. Board eligible. Group, partnership. Available July 1979.

RHEUMATOLOGY—Carlos Ganuza, M.D., 2D Beaver Court, Wilkes-Barre, PA 18702. University of El Salvador 1971. Subspecialty, internal medicine. Board eligible (IM). Institutional, group. Available July 1979.

Stuart P. Brogadir, M.D., 6357 Lancaster Avenue, Philadelphia, PA 19151. Cornell 1975. Subspecialty, internal medicine. Board eligible (IM). Research, group. Available July 1979.

SURGERY, CARDIOVASCULAR—Michael L. Weinberger, M.D., 5902 Weatherford Road, Fayetteville, NC 28303. SUNY—Downstate 1971. Subspecialty, thoracic surgery. Board eligible (general surgery). Group or partnership. Available June 1979.

SURGERY, COLON/RECTAL—Antoine Sara, M.D., 2745 Birchcrest Drive, SE, Apt. 209, Grand Rapids, MI 49506. Damascus (Syria) 1971. Group, partnership, solo. Available August 1979.

SURGERY, GENERAL—Clementino O. Purisima, M.D., 105 Chadwick Road, Teaneck 07666. Santo Tomas (Philippines) 1969. Board eligible. Group partnership, solo. Available July 1979.

Joel A. Yellin, M.D., 215-16 15th Road, Bayside, New York 11360. Rochester 1973. Board eligible. Partnership, solo, group. Available July 1979.

Glenn A. Pasternack, M.D., 7660 Phoenix Drive, Apt. 1559, Houston, TX 77030. SUNY—Downstate 1974. Board eligible. Partnership, group. Available July 1979.

Marc A. Grossman, M.D., 21140 18th Avenue, Bayside, NY 11360. Einstein 1975. Board eligible. Partnership or group. Available July 1979.

Dilipkumar H. Kakaiya, M.D., Lafayette Towers, Apt. 709, 2040 Lehigh Street, Easton, PA 18042. Grant Medical College (India) 1971. Board eligible. Solo, partnership, group. Available July 1979.

Bassam T. Kashlan, M.D., 65 Cedar Avenue, Apt. D-16, Long Branch 07740. Damascus (Syria) 1972. Board eligible. Partnership, institutional, group. Available July 1979.

Frederick J. Heinle, Jr., M.D., 328 S. Linden Avenue, Pittsburgh, PA 15208. Board eligible. Partnership, group, solo. Available July 1979.

Stephen F. Winer, M.D., Box 633, Wiesbaden, West Germany, APO New York 09220. Wayne 1967. Board certified. Partnership, group. Available January 1980.

Meherwan J. Mistry, M.D., 524 Morris Avenue, Apt. 3-E, Elizabeth 07208. Grant Medical College (India) 1972. Board eligible. Partnership, group, solo. Available July 1979.

Ivan A. Shulman, M.D., 785 Burnett Avenue, Apt. 7, San Francisco, CA 94131. Pittsburgh 1972. Board eligible. Group, partnership. Available July 1979.

Vithal D. Kardani, M.D., 133-52 Avery Avenue, Flushing, NY 11355. Jamnagar (India) 1972. House physician or residency. Available.

Vincent Larkin, Jr., M.D., 7021 Colonial Road, Brooklyn, NY 11209. SUNY-Downstate 1974. Subspecialty, vascular surgery. Board eligible. Group or partnership. Available July 1979.

Geoffrey Simon, M.D., RFD 2, Box 155, Harrison, Maine 04040. Boston 1968. Board certified. Group, partnership, solo. Available.

K. Venkataraman, M.D., 380 North Broadway, Apt. C-14, Yonkers, NY 10701. Madras (India) 1966. Board eligible. Solo or group. Available July 1979.

Manuel V. Sarroca, M.D., 300-C Outer Belle Road, Trotwood, OH 45426. University of Philippines 1972. Board eligible. Solo, partnership, group. Available July 1979.

Wei H. Lee, M.D., 3001 S. Harrison Street, Denver, CO 80210. Taipei (Taiwan) 1966. Subspecialty, cardiovascular and thoracic surgery. Any type practice. Available July 1979.

SURGERY, ORTHOPEDIC—Stefan Glowacki, M.D., 2130 Williamsbridge Road, Bronx, NY 10461. Warsaw (Poland) 1962. Subspecialty, orthopedic surgery. Board eligible (orth. surg.) Solo, partnership, research. Available July 1979.

Ronald J. Leavitt, M.D., 717 Willow Way, Narberth, PA 19072. Columbia 1971. Subspecialty, hand surgery. Board certified. Partnership, group. Available July 1979.

Samir R. Dawoud, M.D., 1120B Easton Avenue, Bldg. 18, Somerset 08873. Kasr El Aini (Egypt) 1960. Board eligible. Partnership, group. Available July 1979.

Sheroo Kohli, M.D., 5021 Aurora Avenue, Des Moines, Iowa 50310. G. S. Medical College (India) 1946. Board eligible. Group practice preferred. Available.

SURGERY, UROLOGICAL—Robert J. Claire, M.D, 111 Annadale Road, Staten Island, NY 10312. Guadalajara (Mexico) 1974. Special interest in family counseling. Any type practice. Available July 1980.

Natvarlal P. Patel, M.D., 404 Sixth Stteet, Apt. 5D, Coraville, IA 52241. B. J. Medical School (India) 1970. Subspecialty, general surgery. Board eligible. Solo, partnership, group, research. Available July 1979.

Daniel H. Piazza, M.D., 741 Peninsula Court, Ann Arbor, MI 48105. Illinois 1972. Board eligible. Group or partnership. Available July 1979.

Sheldon Muhlbauer, M.D., 3450-23 Wayne Avenue, Bronx, NY 10467. Einstein 1973. Board eligible. Partnership, group. Available July 1979.

Shree Subhash, M.D., 8 Sugarloaf Court, Baltimore, MD 21209. Prince of Wales Medical College (India) 1967. Partnership, group, solo. Available September 1979.

UROLOGY—H. Barry Opell, M.D., 1275 East 51st Street, Brooklyn, NY 11234. Lausanne (Switzerland) 1971. Board certified. Solo, partnership, group. Available.

Manoj P. Khandherja, M.D., 2901 S. King Drive, Apt. 1818, Chicago, IL 60616. R. G. Kar Medical College (India) 1972. Board eligible. Any type practice. Available July 1979.

Pronouncement of Death at a Home, Medical, or Non-Medical Facility

Amendment to Board of Medical Examiners' Rule NJAC 13:35-6.5

Edwin H. Albano, President of the Board of Medical Examiners in the Division of Consumer Affairs of the Department of Law and Public Safety, pursuant to authority of N.J.S.A. 45:9-1, *et seq.*, hereby adopts the following amendments (indicated by italics) concerning the pronouncement of death at a home, medical, or non-medical facility.

The full text of the rule follows:

13:35-6.5 Pronouncement of death at a home, medical, or non-medical facility

(a) In the event of a death at a home, *medical,* or a non-medical facility, an official pronouncement of the death shall be the primary responsibility of the attending physician or [his] designated substitute.

(b) Upon notification of a probable death *at a home or non-medical facility,* the attending physician or [his] designated substitute shall proceed without inordinate delay to the location of the presumed decedent and make the proper determination and pronouncement of the death. In no case shall a physician direct a funeral director or [his] representative to remove the presumed decedent from the premises until an official pronouncement of the death has been made. *In the case of any death, whether at a home, medical or non-medical facility, the death certificate shall be completed and signed by the attending physician or designated substitute within a reasonable period of time not to exceed 24 hours after the pronouncement of death.*

(c) *The County Medical Examiner,* [I] in cases of death within [the] *such* jurisdiction, [he] shall without inordinate delay require the proper and established means for the pronouncement of the death, arrange for the removal of the body and completion of the death certificate.

(d) [A physician who fails to comply with this rule shall be subject to disciplinary sanction in accordance with the Medical Practice Act.] *Any violation of the foregoing rule by a licensee or any person, association or corporation may be considered as the basis for disciplinary proceedings and sanctions pursuant to N.J.S.A. 45:1-14, 45:9-1, 45:9-22 and 45:9-26.*

(Filing date—February 27, 1979—Subchapter 6 shall expire on October 1, 1983)

Prescribing, Administering or Dispensing Amygdalin (Laetrile)

Board of Medical Examiners' Rule NJAC 13:35-6.17

Edwin H. Albano, President of the Board of Medical Examiners in the Division of Consumer Affairs of the Department of Law and Public Safety, pursuant to authority of N.J.S.A. 45:9-1, *et seq.*, hereby adopts the following regulation concerning the prescribing, administering, or dispensing of amygdalin (laetrile).

The full text of the new rule follows:

13:35-6.17 Prescribing, administering or dispensing amygdalin (laetrile)

(a) The prescription or administration of amygdalin (laetrile) is a medical procedure which may only be performed by a physician licensed to practice medicine and surgery in the State of New Jersey, or a physician duly licensed to practice medicine and surgery in another state provided the practitioner does not open an office or place for the practice of his profession in this State (N.J.S.A. 45:9-21c).

(b) A licensed physician may prescribe, administer or dispense amygdalin (laetrile) to a patient under his care consistent with the following standards and providing that the patient has signed the "written informed request . . . for medical treatment" as set forth herein:

1. Generally:
 i. Either:
 (1) As an adjunct to recognized, customary, or accepted modes of therapy; or
 (2) Utilized exclusively in the treatment of any malignancy, disease, illness or physical condition; and
 ii. If and when the physician has received a confirmed diagnosis of said malignancy, disease, illness or physical condition;
2. In the course of medically justifiable dietary supplement therapy;
3. As a prophylactic medication.

(c) The form concerning the informed request for prescription of laetrile for medical treatment is as follows:

Informed Request for Prescription of Laetrile for Medical Treatment

Patient's name.....
Address.....
Age.....Sex
Name and address of prescribing physician
.....
Malignancy, disease, illness or physical condition diagnosed for medical treatment by amygdalin (laetrile)
.....
My physician has explained to me:

(a) That the manufacture and distribution of amygdalin (laetrile) has been banned by the Federal Food and Drug Administration.

(b) That neither the American Cancer Society, the American Medical Association, nor the Medical Society of New Jersey recommend use of amygdalin (laetrile) in the treatment of any malignancy, disease, illness or physical condition.

(c) That there are alternative recognized treatments for the malignancy, disease, illness or physical condition from which I suffer which he has offered to provide for me including:
(Here describe)
.....

That notwithstanding the foregoing, I hereby request prescription and use of amygdalin (laetrile) (a) in the medical treatment of the malignancy, disease, illness or physical condition from which I suffer [], (b) as a dietary supplement [], or (c) as a prophylactic medication [] Check (a), (b) or (c).
Patient or person signing for patient
.....
Attest:
.....
Prescribing Physician
License No.....
Date.....

A. The above form shall be prepared in quadruplicate and distributed as follows:

(1) Original copy to State Department of Health;

(2) Copy to be retained by the physician;

(3) Copy to patient or person who signed form for the patient;

(4) Copy to pharmacist.

B. When amygdalin (laetrile) is utilized in the treatment of a malignancy, the diagnosis of malignancy shall be documented by a positive tissue diagnosis rendered by a qualified pathologist which shall include the size, location and type of malignancy. In the absence of tissue for diagnosis, the treating physician shall be required to obtain consultative and/or professional reports to support a positive diagnosis of a

malignancy.

C. The alternative medically recognized and accepted form of therapy offered by a physician shall be thoroughly discussed with the patient and documented in writing.

(d) Complete and accurate records shall be maintained and made available to include:

1. Copy of signed informed request.

2. History of previous and current therapy to be included where indicated.

i. Surgery;

ii. Radiation;

iii. Chemotherapy.

3. Complete record of dates of office visits, examination, and evaluation of patient with detailed progress notes.

i. Complications and/or untoward reactions from amygdalin (laetrile) shall be reported immediately to the State Department of Health.

ii. Fee for service: The patient record shall include fee charged per visit which fee shall not be greater than the physician's usual and customary fee for an office visit. When fee includes administering or dispensing amygdalin (laetrile), the charge is to be itemized and recorded. When a physician administers or dispenses amygdalin (laetrile), the fee to the patient shall not exceed the cost to the physician of such substance and shall be so itemized in the charge or billing.

iii. Copies of all laboratory and follow-up examinations, and

iv. Periodic clinical measurements of tumor activity.

4. Date of procurement of amygdalin (laetrile), quantity, cost, name and address of manufacturer and supplier, batch number and expiration date when administered or dispensed by a physician.

5. Records are to be readily available without prior notice for inspection by the appropriate official agency, including but not limited to the New Jersey State Board of Medical Examiners and the New Jersey State Department of Health.

6. Copies of records shall be forwarded to State Department of Health at quarterly intervals.

(e) Solicitation is prohibited. Such prohibited activity shall include, but is not limited to, the dissemination of information concerning amygdalin (laetrile) which may be found by the Board of Medical Examiners as:

1. False, fraudulent, deceptive, misleading or flamboyant;

2. Using testimonials;

3. Guaranteeing that satisfaction or cure will result from the use of amygdalin (laetrile);

4. Making claims of professional superiority;

5. Stating fees for professional services which are false, deceptive and/or misleading.

(f) A licensed physician may, in the regular course of medical practice and pursuant to a justifiable medical basis, prescribe, administer, or dispense amygdalin (laetrile) in accordance with the Act concerning Laetrile (Chapter 318, P.L. 1977) and these rules and regulations.

(g) Any violation of the foregoing rule may be considered unprofessional conduct, professional incompetence, gross malpractice and/or gross neglect in the practice of medicine and provide grounds for suspension or revocation proceedings being instituted against the licensee.

(Filing date—March 5, 1979—Subchapter 6 shall expire on October 1, 1983)

Uses of Amphetamines and Sympathomimetic Amines

Board of Medical Examiners' Rule NJAC 13:35-6.16

Edwin H. Albano, President of the Board of Medical Examiners in the Division of Consumer Affairs of the Department of Law and Public Safety, pursuant to authority of N.J.S.A. 45:9-1, *et seq.*, hereby adopts the following new regulations concerning the prescribing, administering, and dispensing of amphetamines and sympathomimetic amines.

The full text of the new rule follows:

13:35-6.16 Uses of amphetamines and sympathomimetic amine drugs

(a) No physician shall prescribe, order, dispense, administer, sell or transfer any amphetamine or sympathomimetic amine drug or compound designated as a Schedule II Controlled Dangerous Substance pursuant to the laws of New Jersey, to or for any person except:

1. for the treatment of:

i. narcolepsy

ii. hyperkinesis

iii. drug-induced brain dysfunction

iv. epilepsy

v. depression shown to be refractory to other therapeutic modalities; or

2. for the differential diagnostic psychiatric evaluation of depression; or

3. for the clinical investigation of the effects of such drugs or compounds in which case, in addition to other requirements of applicable laws, prior application therefor shall have been made to the Board of Medical Examiners and approval granted before any such investigation is begun.

(b) in addition to the prohibitions set forth in subparagraph (a) hereinabove, no physician shall prescribe, order, dispense, administer, sell or transfer any amphetamine or sympathomimetic amine drug or compound designated as a Schedule II Controlled Dangerous Substance pursuant to the laws of New Jersey, for use in weight management, dieting or any other anorectic purpose, or for the treatment of fatigue.

(c) Violation of any of the foregoing may be deemed to constitute one or more of the following:

1. distribution or dispensing of a controlled dangerous substance in an indiscriminate manner, not in good faith, or without good cause, pursuant to N.J.S.A. 45:1-13; or

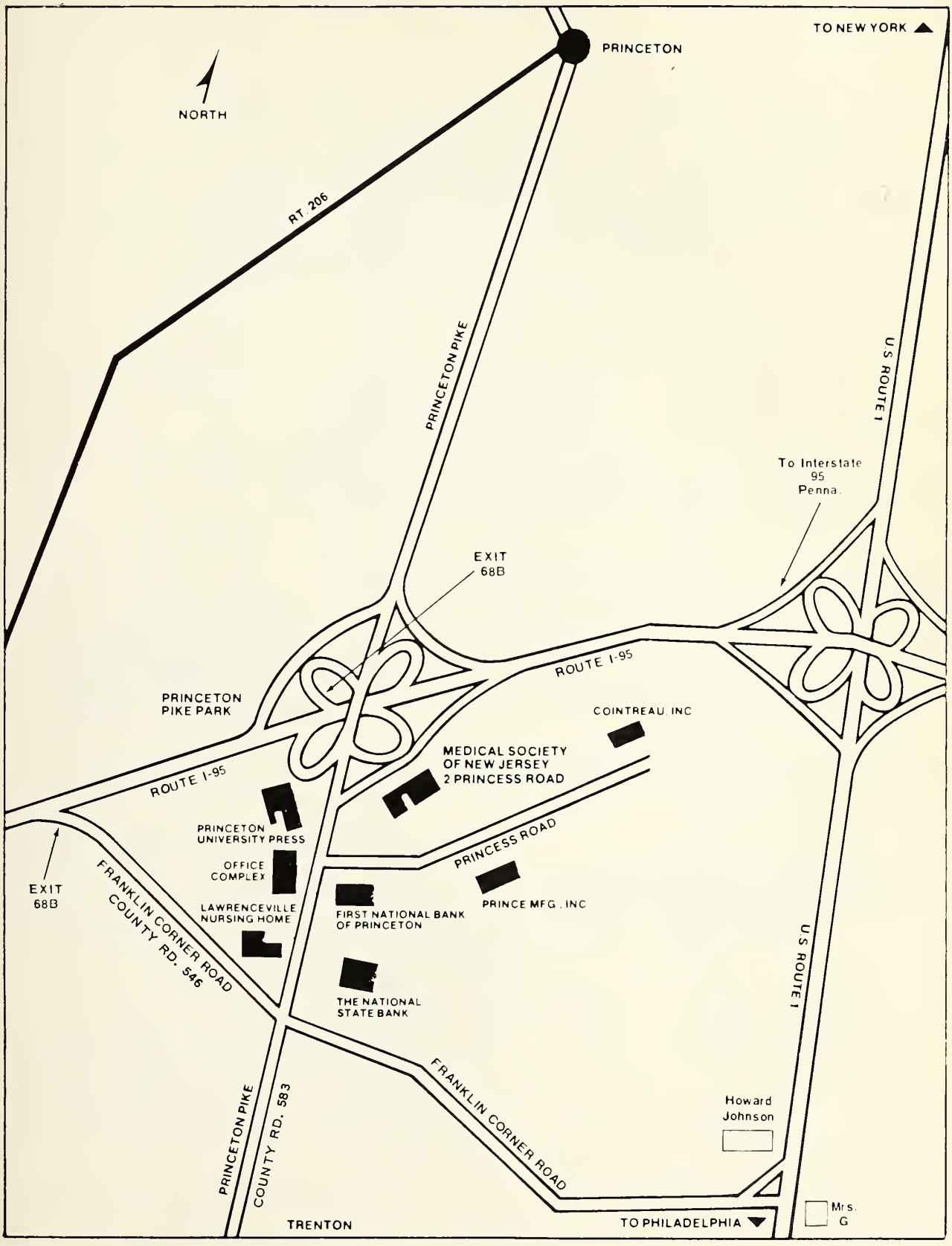
2. gross malpractice, gross neglect, or gross incompetence in the practice of medicine, pursuant to N.J.S.A. 45:1-21(d); or

3. professional misconduct in the practice of medicine, pursuant to N.J.S.A. 45:1-21(e).

(d) The following list, although not exhaustive or exclusive, does include many of the generic and brand-name Schedule II drugs available as of September 1, 1978, which fall within the above regulation: amphetamine, Benzedrine, Biphetamine, Desoxyn, Dexamyl, Dexedrine, dextroamphetamine, Eskatrol, Fetamin, methamphetamine, methylphenidate, Obetrol, Obotan, phenmetrazine, Preludin and Ritalin.

(Filing date—March 21, 1979—Subchapter 6 shall expire on October 1, 1983)

Area Map—MSNJ Headquarters



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SEPTEMBER 13-16, 1979

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CME CALENDAR

This listing is compiled through the cooperation of the Committee on Medical Education of the Medical Society of New Jersey, the Academy of Medicine of New Jersey, the New Jersey Chapter of the American Academy of Family Physicians, and the Office of Continuing Medical Education of the College of Medicine and Dentistry of New Jersey. For information on accreditation, please contact the sponsorings organization(s), indicated by italics—last line of each item.

MEDICINE (includes Family, Internal, and General Medicine and Dermatology)

June

20 **Advances in Medicine**
9:30 a.m.-11 a.m.—Bergen Pines County Hospital, Paramus
(Bergen Pines County Hospital and AMNJ)

20 **Medical Lecture Series**
27 1-3 p.m.—Christ Hospital, Jersey City
(Christ Hospital and AMNJ)

21 **Blood Disease Tests**
28 **Drug Interactions**
11:45 a.m.-12:45 p.m.—John F. Kennedy Medical Center, Edison
(John F. Kennedy Medical Center)

21 **Cardiology Conferences**
28 2:15-4:15 p.m.—Deborah Heart and Lung Center, Browns Mills
(Deborah Heart and Lung Center and AMNJ)

21 **Regional Chest Conferences**
7:30-9:30 p.m.—Newark Beth Israel Medical Center
(NJ Thoracic Society and AMNJ)

21 **Infectious Diseases**
4-6:30 p.m.—Somerset Hospital, Somerville
(Somerset Hospital and AMNJ)

26 **Scanning, New Developments**
8-9 p.m.—Warren Hospital, Phillipsburg
(Warren Hospital and AMNJ)

26 **Weekly Tumor Conferences**
11 a.m.-12 noon—Morristown Memorial Hospitla
(Morristown Memorial Hospital and AMNJ)

27 **Cardiac Grand Rounds**
3-4:30 p.m.—CMDNJ-NJ Medical School
(American Heart Association, NJ Affiliate and AMNJ)

July

5 **Pharmacokinetics and Pharmacodynamics, Part I**

12 **Pharmacokinetics and Pharmacodynamics, Part II**
12 noon-1 p.m.—Carrier Foundation, Belle Mead
(Carrier Foundation)

13-15 **ECG Interpretation and Arrhythmia Management**
Playboy Resort, Great Gorge, McAfee
(International Medical Education Corp. and AAFP)

Aug.

IgE in Allergic Disease
2 7:30 a.m.-5 p.m.
3 8 a.m.-5 p.m.
4 8 a.m.-4:30 p.m.
5 8-12 noon
Downingtown Inn, Downingtown, Pa.
(Holy Name Hospital and AMNJ)

NEUROLOGY/PSYCHIATRY

June

20 **Sexual Medicine**
1:30-3 p.m.—VA Medical Center, Lyons
(VA Medical Center and AMNJ)

20 **Behavioral and Mental Disorders**
5-9 p.m.—Mountainside Hospital, Montclair
(Mountainside Hospital)

July

19 **Twilight Zone**

26 **Phenomenology in Psychiatric Differential Diagnosis, Part I**
12 noon-1 p.m.—Carrier Clinic, Belle Mead
(Carrier Foundation)

Aug.

2 **Phenomenology in Psyhiatric Differential Diagnosis, Part II**

9 **Role of the Hypothalamus in Health and Disease**

16 **Therapeutic Community**

23 **Hyperactive Child Syndrome**

30 **Depression**
12 noon-1 p.m.—Carrier Foundation, Belle Mead
(Carrier Foundation)

OBSTETRICS/GYNECOLOGY

June

20 **Grand Rounds and Case Presentations**
27 2-4 p.m.—Rotating between Martland Hospital, Newark Beth Israel, St. Michael's, and St. Joseph's Hospitals, Newark, and Jersey City Medical Center
(CMDNJ and AMNJ)

21 **Grand Rounds and Case Presentations**
28 4-5 p.m.—Martland Hospital, Newark
(CMDNJ and AMNJ)

PATHOLOGY/CLINICAL PATHOLOGY

June

20 **Clinical Pathology Grand Rounds**
12 noon-1 p.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

27 **Neuropathology Conferences**
8-9:15 a.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

SURGICAL SPECIALITIES (includes ENT, neurosurgery, ophthalmology, orthopedic, plastic, and vascular surgery)

June

20 **Management of Face Turns with Nystagmus**
7:15-9:30 p.m.—United Hospitals Medical Center, Annex Bldg., Newark
(Associated Eye Residencies of NJ and AMNJ)

21 **Neurosurgical Case Presentations**
4-30 p.m.—NJ Medical School, Newark
(CMDNJ and AMNJ)

22 **Orthopedics**
7:30-9 a.m.—Alexian Brothers Hospital, Elizabeth
(Alexian Brothers Hospital

26 **Cervical Spine Injuries**
6-10 p.m.—Englewood Club, Englewood
(Englewood Surgical Society and AMNJ)

MISCELLANEOUS

June

26 **PSRO**
11 a.m.-12 noon—Greystone Park Psychiatric Hospital
(Greystone Park Psychiatric Hospital and AMNJ)

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LETTERS TO THE JOURNAL

More on Generic Substitution

January 25, 1979

Dear Sir:

The December, 1978 editorial (JMSNJ 74:895) concerning generic substitution interested us greatly. While the writer is to be commended for his careful exposition of the generic substitution law's specific provisions, a few factual inaccuracies need correction.

In addition, his opinions on the ramifications of the law demand rebuttal.

The factual errors include:

1. The writer stated that the generic substitution law has been in effect since September 29, 1977.

Fact: The law was enacted on that date, but it is *not* yet in effect, nor will it be until the list of interchangeable drug products has been prepared and distributed by the Drug Utilization Review Council (DURC).

The Drug Utilization Review Council has adopted a very conservative approach to the list which accounts for the delay in the law's implementation.

2. The writer stated ". . . there are only seventeen states which have anti-substitution laws at present."

Fact: Fewer than ten states have retained their anti-substitution laws.

3. The writer stated that the pharmacist's legal liabilities are "greatly enhanced" under generic substitution laws.

Fact: The New York State Assembly's Office of Legislative Oversight and Analysis conducted legal research, discussion with insurance companies and communication with major drug manufacturers on this issue. It was concluded that the allegations of increased liability for pharmacists and physicians under generic substitution laws "have no basis in fact, . . . are malicious and cleverly designed to frighten the pharmacists and physicians into prescribing high-priced trade name products when lower-priced and equivalent generics are available."[1]

The opinions expressed about the effect of generic substitution on the pharmaceutical manufacturer were so interwoven with non-germane issues, such as FDA regulations, the role of counterfeiters, and references to "Italian tetracycline" that the reader probably couldn't discern the real issue. The real issue is, should the patient, pharmacist, and physician believe the FDA's contention that all medications are subject to the same manufacturing criteria or should they believe the pharmaceutical industry's contention that only "research-intensive" companies can make products well?

The editor is obviously sympathetic to the brand-name manufacturer's contentions. He states that the generic manufacturer's major motive is profit, while claiming only exalted motivation for "respected major pharmaceutical manufacturers." These same major manufacturers have consistently shown a rate of return on equity averaging 63% *higher* than that of other manufacturers.[2] Apparently, they may have some profit motivation also.

The editor's concern about possible decreases in research spending is one often voiced. Is it not more reasonable to expect that research for new, patentable products for which manufacturers can extract high profits for the life of the patent will be increased to offset decreased earnings on products for which the patent has expired?

Is generic substitution really something new? Nationally, one in eight prescriptions is written generically, allowing the pharmacist to use *any* company's product to fill the prescription.[3] These generic products aren't inconsequential in nature, either. They include prednisone, digoxin, nitroglycerin, and quinidine. Why physicians would allow *any* company to be used for one in eight prescriptions, yet become concerned about quality issues when legislators attempt to increase the percentage so as to allow expanded savings, eludes us.

Our response to the question, "Will the generic substitution laws save money for the consumer?" is "yes"—if physicians and pharmacists keep an open mind and allow the laws to work, and if patients are informed about their choices in an unbiased manner.

As the article pointed out, the physician's option to negate substitution reserves his right to specify the exact drug product. Rather than exercising that option in a uniformly negative fashion, as advised by the editorial, physicians should selectively choose that option when medically necessary for an individual patient. By doing so, the law will not be thwarted and the patient can receive both effective and economically sound drug therapy.

(signed) Sanford Luger, R.P., Chairman
Thomas T. Culkin, PharmD, MSc.
Executive Director
Drug Utilization Review Council

[1]Special report to Assemblyman H. L. Strelzin, July 25, 1978.
[2]*The Pharmaceutical Industry*, C. M. Lindsay (ed) Wiley, 1978.
[3]*Pharmacy Times*, April 1978, p. 42.
[4]See Commentary, p. 458, this issue

Watch for the Danger Signs

March 12, 1979

Dear Editor:

Having been born and raised in a country where there was no "Bill of Rights," no freedom of speech for the people, I am worried that Americans are giving their freedom away to an ever-growing bureaucratic system.

As a student of history, as an adopted American, I know I have rights and I cherish the freedom which the Constitution provides me. I do not feel that too many regulations will make life better for you and me as individuals. Knowing the dark sides of other government systems, do not ignore the danger ahead if we over-regulate our lives, diminish our freedom of choices and ignore a voluntary system which made this country

great and exists still in New Jersey in the voluntary ambulance squads, fire departments, and other organizations.

Now we see the State Department of Health introducing more regulations, regulations that can destroy volunteerism in New Jersey. Such a shame.

Power and money seem to be more important today than freedoms, than rights and even justice.

Yes, I am afraid when I see those same danger signs today, some thirty years later, and people not paying attention to history.

I feel it was and is a privilege to be an American, to have given many hours of my life without compensation to this State and the people through the voluntary sector.

If we continue to allow governmental agencies to make regulations without even a public hearing, what will be left?

(signed) Rudolf E. Schwaeble, M.D.

Business Is Flexing Its Biceps

March 13, 1979

Dear Sir:

In 1977, $34 billion were spent by industry toward the payment of health insurance, including major medical. This did not include all the out-of-pocket costs for ambulatory care. Business feels that the long-term controls of these costs are very slim, unless it tries to influence and alter the health benefits program, and works to influence the health care system in its respective communities. It strongly favors a businesslike approach to an improved health care system and an approach that requires the least amount of public intervention and regulation.

A recent study titled, "A National Health Care Strategy," was conducted by the National Chamber Foundation for the United States Chamber of Commerce in Washington, D. C. The study was completed in October 1978, and many important and pertinent findings are included in the report. A summary of a main portion of the report contains the following:

1. Comprehensive insurance and third-party reimbursement arrangements greatly have decreased both consumer and provider "price consciousness," with resultant cost escalation at *nearly double the overall inflation rate.* The market-oriented solution to this problem and the one that can be achieved *through private sector initiatives* is stimulation of *competition* within the health care delivery system.

2. Competition is the approach most likely to produce long-term cost containment because it addresses the cause of the problem—the flawed economic structure of the health care system—rather than only its symptoms. The proliferation of *alternate health care delivery systems* can create the needed competition.

3. A form of alternate health care delivery system is an organization in which a *specific set of providers* assumes responsibility for delivery of health care services to *voluntarily enrolled members* in exchange for a *fixed premium.* This is in contrast with the traditional system of health care delivery in which the consumer seeks care from a variety of providers and must pay all costs not covered by insurance.

4. The majority of currently operating alternate health care delivery systems are known as HMOs. Successful HMO operation depends on responsible physician involvement in the total HMO operation, including its financing; in many HMOs, providers accept some degree of risk for the provision of services.

5. HMOs contain costs by substituting less expensive modes of care (office visits) for more expensive care (inpatient hospitalization) wherever possible, and by stressing preventive measures, as well as treatment. As a result, HMOs *can decrease hospital use as much as 50 percent.* Because HMOs rely less on costly, rapidly inflating hospital care, premiums tend to rise more slowly than traditional health insurance premiums. In some areas HMO rates are priced below the cost of comprehensive health insurance and, therefore, represent a real challenge to the traditional health care system.

6. Competition for enrollees between the HMOs and the traditional health insurers can result in better controlled health care costs in an entire community. There are areas where this has occurred, primarily because *businesses have helped foster the necessary competition.*

7. Businesses pay a large proportion of total personal health care costs. Federal and state laws now obligate many businessess to offer HMOs to employees under certain conditions.

8. The business community appears more likely to succeed in encouraging competition in the health care field than the federal government.

Heretofore, most of us felt that the "Government" was the "whipping boy." Now it becomes apparent that business is flexing its biceps. It appears wiser that we as physicians make the necessary inroads to meet the challenge of the day, rather than have others do it for us in such a way as to disrupt an existing system of medicine. Competition is the order of the day and it is best not to minimize it.

John C. Stockman, M.D.

Helping Smokers Quit

April 2, 1979

Gentlemen:

A recent study indicates that many smokers would try to quit if their physicians told them to. However, about two-thirds of smokers report that they never have received advice from their physicians on quitting. We believe some doctors don't tell their patients to quit smoking for the reason that they don't know how to answer the inevitable "how."

To help physicians encourage their patients to stop smoking, the National Cancer Institute has developed the "Helping Smokers Quit" kit. (An explanatory folder which describes the concept and materials included is available from the Office of Cancer Communications, National Cancer Institute, Bldg. 31, Room 4B39, Bethesda, MD 20014.) Available free of charge, the kit contains enough material to assist fifty smokers who want to stop.

Response has been very positive. To date, over 25,000 kits have been ordered by interested health professionals, and the program has received support in the journals and newsletters of the American Medical Association, American Academy of Family Physicians, American College of Obstetricians and Gynecologists, the National Medical Association, and several state medical societies.

You can order your kit directly by writing to "Helping Smokers Quit," Dept. K-64, National Cancer Institute, Bethesda, MD 20205. We believe this material can make a major contribution to your efforts to prevent cancer and other chronic diseases among your patients.

(signed) Bernard J. Ellis, Jr.
Program Director
Office of Cancer Communications
National Cancer Institute

Caution on Treatment for Gonorrhea in Pregnancy

April 21, 1979

Dear Doctor Krosnick:

I am presently a Senior Fellow in Maternal-Foetal Medicine at Columbia-Presbyterian Medical Center, Babies Hospital in New York.

In the April issue of your journal, there was an article concerning the "Treatment Schedules for Gonococcal Infections—1978"—*J Med Soc NJ* 76:306-308. I take exception with the treatment stated for gonorrhea in pregnancy. Spectinomycin *should not* be given to a pregnant patient who is allergic to penicillin.

Spectinomycin is produced by *Streptomyces spectabilis*. This is in the same class as the tetracyclines which are elaborated and produced by *Streptomyces aureofaciens*. As we all know, the tetracyclines are notorious for brown discolorations of the teeth and deposition of the drug in the teeth and bones. This is due to the chelating property of and the formation of a tetracycline-calcium orthophosphate complex.

A better alternative form of treatment is erythromycin given orally, i.e., 2 grams stat and the 500 milligrams, four times a day for five days, resulting in a total dose of twelve grams.

I know this article was compiled from the information given to you by the Center for Disease Control. Perhaps the authors may now correct their original source.

Thank you for your time and consideration in this matter.

(signed) Yvonne S. Thornton, M.D.

OBITUARIES

Dr. Paul K. Ayars

Word has just been received that Paul K. Ayars, M.D., a member of our Cumberland County component, died on January 9, 1979. A native of Philadelphia, born in 1918, Dr. Ayars earned his doctorate of medicine from Hahnemann Medical College in Philadelphia in 1947. He had practiced general medicine in Port Norris for many years and was affiliated with the Bridgeton Hospital. He was a Fellow of the American Academy of Family Physicians. Dr. Ayars was active in medical affairs and had been Reporter and President of his county medical society (1957-1958). During the Korean conflict he served with the medical department of the United States Navy.

Dr. Frank D. Bellucci

A member of our Essex County component, Frank D. Bellucci, M.D., died on March 24. Born in 1914, Dr. Bellucci received his medical degree from the University of Bologna (Italy), class of 1940, and established a practice in obstetrics and gynecology in Newark. He was a Fellow of the American College of Obstetricians and Gynecologists, and had been affiliated with Columbus Hospital in Newark. Dr. Bellucci served for three years with the United States Public Health Service during World War II.

Dr. Edward T. Cicione

Edward Thomas Cicione, M.D., a member of our Burlington County component, died on April 14, after a prolonged illness. Born in 1914, Dr. Cicione was graduated from Hahnemann Medical College in 1941 and practiced general medicine in Riverton and Palmyra for ten years before pursuing a residency in obstetrics and gynecology at the University of Pennsylvania, after which he established offices for the practice of those specialties in Camden. He was a Fellow of the American College of Obstetrics and Gynecology and had been affiliated with Our Lady of Lourdes Hospital. During World War II, Dr. Cicione served in the department of medicine of the Army of the United States.

Dr. Edwin C. Greene

Edwin C. Greene of Bridgeton died on March 7 after a short illness. Born in 1912 in Alfred, New York, Dr. Greene received his medical degree from George Washington University School of Medicine, class of 1938, and pursued a residency in general medicine at Gallinger Municipal Hospital in Washington, D.C. He practiced family medicine, with special interest in obstetrics in Cumberland County and was associated with the Bridgeton General Hospital. He was active in organized medicine and served as President of his county medical society (Cumberland) in 1951-1952. During World War II, Dr. Greene was on active duty with the medical department of the AUS.

Dr. John Huberman

Word just has been received of the death on August 13, 1978, of John Huberman, M.D., a senior member of our Essex County component. Born in Russia, Dr. Huberman emigrated to the United States and earned his degree in medicine from New York University's Medical School graduating in 1908. Prior to his retirement to Hallandale, Florida, Dr. Huberman practiced gynecology in Newark, and was a Fellow of the American College of Surgeons, the International College of Surgeons, and the American College of Obstetricians and Gynecologists. He had affiliations with Doctors Hospital and Newark Beth Israel Medical Center in Newark, and had been the recipient of a number of awards during his career. He had received MSNJ's Golden Merit Award in 1958, commemorating his fifty years of practice in the medical profession; and was honored that same year by the Internes' Alumni of Newark Beth Israel Medical Center both for his fifty years of service to that institution, and for being the first intern to serve there.

Dr. Huberman also distinguished himself in the field of medical research on hormones, using Squibb Company's laboratory and research facilities at their invitation. He is survived by a son, Victor Huberman, M.D., also a graduate of NYU's medical school and internship at Newark Beth Israel Medical Center, and by two grandsons, Mark Leibo, M.D. and Herbert Lipschutz, M.D., alumni of these same institutions. Dr. Huberman was 92 years old at the time of his death.

Dr. Paul T. Milnamow

On March 15, Paul T. Milnamow, M.D., formerly of Camden, died at his home after a lengthy illness. Born in 1913 in Wilkes Barre, Pennsylvania, Dr. Milnamow was graduated from Hahnemann Medical College in 1940 and came to Camden for the practice of general surgery. He was affiliated with West Jersey Hospital there. Later his offices were moved to Philadelphia and he was on the staff at Saint Luke and Children's Medical Center in the practice of industrial and general surgery. He was a Fellow of the International College of Surgeons. During World War II and again during the Korean conflict, Dr. Milnamow served in the Medical Department of the Army of the United States.

Dr. Jessie D. Read

Word has been received of the death on December 29, 1978 of Jessie D. Read, M.D., a member of our Union County component, who had practiced in Westfield and Newark. A graduate of Long Island University College of Medicine, class of 1928, Dr. Read pursued a residency in obstetrics and gynecology at Bellevue Hospital College of Medicine. She was board certified in her chosen field, a Founding Fellow of the American College of Obstetricians and Gynecologists, and a Fellow of the American College of Surgeons, and had been affiliated with St. Michael's Medical Center in Newark and St. Barnabas Medical Center in Livingston, ultimately becoming chief of the department of obstetrics and gynecology at the latter institution. During World War II, Dr. Read served with the medical department of the Army of the United States. She was a 1978 recipient of MSNJ's Golden Merit Award, indicating fifty years of medical practice.

Dr. Joseph D. Ruggio

We just have learned of the death on February 7 of Joseph D. Ruggio, M.D., at Newark Beth Israel Medical Center. A member of our Essex County component, Dr. Ruggio was born in 1917 in New York City, and graduated from Creighton Medical School in 1943. He established a practice in general and internal medicine in North Arlington and had been staff physician at North Arlington High School and for the Newark draft board during World War II. Dr. Ruggio also was affiliated with West Hudson Hospital in Kearny.

Dr. Herbert Steiner

Herbert Steiner, M.D., of Irvington, died on February 25 at his home, at the venerable age of 87. Born in Berlin, Germany, Dr. Steiner earned his medical degree at the University of Vienna in Austria, graduating in 1916. He emigrated to the United States, and for 41 years was a general practitioner in Irvington. Dr. Steiner was affiliated with United Hospitals of Newark, Newark Beth Israel Medical Center, and Irvington General Hospital. He had been a member of the New Jersey Obstetrical and Gynecological Society.

Dr. D. Blair Sulouff

One of Morris County's senior members, D. Blair Sulouff, M.D., died on April 19. Born in New York City on New Year's Day, 1908, Dr. Sulouff earned his medical degree from the University of Pennsylvania School of Medicine, class of 1933, and went on to pursue a career in ophthalmology. He was affiliated with St. Clare's Hospital, Denville, Morristown Memorial Hospital, and All Souls Hospital, Morristown, where he was chief of the department of opthalmology. Dr. Sulouff had been active in Medical Society affairs and served as a member of the Committee on Conservation of Vision from 1967 to 1974. He had been a member of the Academy of Medicine of New Jersey and the New Jersey Academy of Ophthalmology and Otolaryngology. During World War II, Dr. Sulouff served in the department of medicine of the Army of the United States.

Dr. Charles B. Whitlock

Word has been received of the untimely death on February 9 of Charles Barton Whitlock, M.D., of Montclair. Dr. Whitlock had been an ophthalmologist in that community for 20 years. He was born in Memphis, Tennessee, and received his medical degree from George Washington University, class of 1954. He served his internship at the U.S. Naval Hospital, Jacksonville, Florida, and took graduate studies in ophthalmology and Columbia-Presbyterian Medical Center. Dr. Whitlock was a staff member at Mountainside and Community Hospitals in Montclair. He was 49 years old at the time of his death.

Dr. Asher Yaguda

An emeritus member of our Essex County component, Asher Yaguda, M.D., died on November 17, 1978. Born in 1897 in Albany, New York, Dr. Yaguda attended Albany Medical College, graduating in 1920. Prior to retirement he practiced pathology and internal medicine in Newark, and had been affiliated with Martland Hospital in Newark. He was a diplomate in clinical pathology and pathologic anatomy, a Fellow of the American College of Physicians, and a Fellow of the College of American Pathologists. Dr. Yaguda was active in medical affairs in his county and at the state level had been a member or chairman of several committees including the Annual Meeting Committee, the Advisory Committee to the Woman's Auxiliary, and the old Welfare Committee and its subcommittees. He was living in Port Jervis, New York at the time of his death.

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